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SOME BYGONE OPERATIONS IN SURGERY.

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III. CUTTING FOR THE STONE *(continued)*.

CHESELDEN'S OPERATION.

JOHANNES JACOBUS RAU (1668–1719) was born in Baden; he was educated at Strasburg, acted for a time as ship's surgeon, and visited Spain and England. He then studied in Leyden and Paris, where he became interested in the discussion on the relative advantages of the different methods of cutting for the stone. Soon afterwards he settled in Amsterdam, where he taught anatomy privately and was appointed operating surgeon to the State Hospital. Finally, in 1713, he succeeded Bidloo as Professor of Medicine, Surgery, and Anatomy at the University of Leyden.

When Frère Jacques was operating in Holland Rau took the opportunity of dissecting the bodies of those who died, and thus learnt that the improved operation consisted in dividing the prostate and neck of the bladder. Knowing this and adopting it with much success he, deliberately and to his lasting shame, concealed the method and taught his pupils that he still cut through the posterior vesical wall, a dangerous operation with a high mortality. Amongst those who were thus taught was Albinus, Rau's favourite pupil and assistant. Albinus in turn taught Morand, the leading surgeon in Paris, and Cheselden in England performed the operation as it was described by Albinus. Both surgeons were profoundly dissatisfied with the results. They complained to Albinus, who, acting in perfect good faith, found that he had himself been deceived.

Cheselden (1688–1752), who was lithotomist to St. Thomas's Hospital from 1723 to 1727, immediately set himself to devise a more satisfactory operation based upon correct anatomical lines. After passing his operation through an experimental stage he thus described the technique:—

"I tie the patient as for the greater apparatus, but lay him upon a blanket several doubles upon an horizontal table about three feet high, with his head only raised. I first make as long an incision as I can cutting down between the musculus accelerator urinæ and erector penis, and by the side of the intestinum rectum. I then feel for the staff, holding down the gut all the while

with one or two fingers of my left hand, and cut upon it in that part of the urethra which lies beyond the corpora cavernosa urethræ and in the prostate gland cutting from below upwards to avoid wounding the gut; and then passing the gorget very carefully in the groove of the staff into the bladder, bear the point of the gorget hard against the staff, observing all the while that they do not separate and let the gorget slip to the outside of the bladder. Then I pass the forceps into the right side of the bladder, the wound being on the left side of the perinæum; and as they pass, carefully attending to their entering the bladder, which is known by their overcoming a straitness which there will be in the place of the wound; then taking care to push them no farther, that the bladder may not be hurt I first feel for the stone with the end of them, which, having felt, I open the forceps and slide one blade underneath it and the other at top and then extract it very deliberately that it may not slip suddenly out of the forceps and that the parts of the wound may have time to stretch, taking great care not to gripe it so hard as to break it. And if I find the stone very large I again cut upon it as it is held in the forceps.

"Here I must take notice it is very convenient to have the bladder empty of urine before the operation; for if there is any quantity to flow out of the bladder at the passing of the gorget, the bladder does not contract but collapses into folds which makes it difficult to lay hold of the stone without hurting the bladder. But if the bladder is contracted it is so easy to lay hold of it that I have never been delayed one moment, unless the stone was very small. Lastly I tie the blood-vessels by the help of a crooked needle and use no other dressing than a little bit of lint besmeared with blood that it may not stick too long in the wound. And all the dressings during the cure are very slight, almost superficial, and without any bandage to retain them because that will be wetted with urine and gall the skin. At first I keep the patient very cool to prevent bleeding and sometimes apply a rag dipt in cold water to the wound and to the genital parts, which I have found very useful in hot weather particularly. In children it is often alone sufficient to stop the bleeding and always helpful in men.

"The day before the operation I give a purge to empty the guts and never neglect to give some laxative medicine or clyster a few days after if the belly is at all tense or if they have not a natural stool. What moved me to try this way, if I may be allowed to know my own mind, was the consideration of women scarce ever dying of this operation from which I concluded that if I could cut into the urethra beyond the corpora cavernosa urethræ the operation would be nearly as safe in men as in women.

"What success I have had in my private practice I have kept no account of because I had no intention to publish it, that not being sufficiently witnessed. Publicly in St. Thomas' Hospital I have cut two hundred and thirteen; of the first fifty only three died; of the second fifty, three; of the third fifty, eight; and of the last sixty-three, six. Several of these patients had the small-pox during their cure some of which died but I think not more in proportion than what usually die of that distemper; these are not reckoned among those who died of the operation. The reason why so few died in the two first fifties was that at that time, few very bad cases offered; in the third, the operation

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being in high request, even the most aged and the most miserable cases expected to be saved by it. One of the three that died out of the one hundred and five was very ill with whooping cough; another bled to death by an artery into the bladder, it being very hot weather at that time. But this accident taught me afterwards, whenever a vessel bled that I could not find, to dilate the wound with a knife till I could see it.

"If I have any reputation in this way I have earned it dearly, for no one ever endured more anxiety and sickness before an operation, yet from the time I began to operate all uneasiness ceased and if I have had better success than some others I do not impute it to more knowledge but to the happiness of mind that was never ruffled or disconcerted and a hand that never trembled during any operation."

This is Cheselden's own account. Sauveur François Morand (1697–1773), son of the chief surgeon to the Hôtel des Invalides in Paris, was sent to England by the Royal Academy of France to report upon the operation in 1729, and states that he often saw a stone removed in twenty-four seconds and that it rarely took more than a minute. He watched Cheselden cut twenty-seven patients without losing one, and spoke so favourably of the method that it soon became known and practised throughout Europe. It remained in ordinary use until 1885, when lithotrity—crushing the stone—came into vogue.

The *Transactions of the Royal Medico-Chirurgical Society* (1819, x, 153) contains an interesting personal account of the operation from the patient's side. It was performed on Dec. 30, 1811, and the anonymous patient says:—

"My habit and constitution being good it required little preparation of body, and my mind was made up. When all parties had arrived I retired to my room for a minute, bent my knee in silent adoration and submission and returning to the surgeons conducted them to the apartment in which the preparations had been made. The bandages &c. having been adjusted I was prepared to receive a shock of pain of extreme violence and so much had I over-rated it, that the first incision did not even make me wince although I had declared that it was not my intention to restrain such impulse, convinced that such effort of restraint could only lead to additional exhaustion. At subsequent moments, therefore I did cry out under the pain, but was allowed to have gone through the operation with great firmness.

"The forcing up of the staff prior to the introduction of the gorget gave me the first real pain, but this instantly subsided after the incision of the bladder was made, the rush of urine appeared to relieve it and soothe the wound.

"When the forceps was introduced the pain was again very considerable and every movement of the instrument in endeavouring to find the stone increased it. Still, however, my mind was firm and confident, and, although anxious, I was yet alive to what was going on. After several ineffectual attempts to grasp the stone I heard the operator say in the lowest whisper 'It is a little awkward, it lies under my hand. Give me the curved forceps' upon which he withdrew the others. Here, I think, I asked if there was anything wrong—or something to that purport—and was reanimated by the reply conveyed in the kindest manner, 'Be patient, Sir, it will soon be over'. When

the other forceps was introduced I had again to undergo the searching for the stone and heard Mr. Cline [1750–1827, Surgeon to St. Thomas's Hospital] say 'I have got it'. I had probably by this time conceived that the worst was over; but when the necessary force was applied to withdraw the stone the sensation was such as I cannot find words to describe. In addition to the positive pain there was something most peculiar in the feel. The bladder embraced the stone as firmly as the stone was itself grasped by the forceps; it seemed as if the whole organ was about to be torn out. The duration, however, of this really trying part of the operation was short and when the words 'Now, Sir, it is all over' struck my ear, the ejaculation of 'Thank God! Thank God!' was uttered with a fervency and fulness of heart which can only be conceived. I am quite unable to describe my sensations at the moment. There was a feeling of release, not from the pain of the operation, for that was gone and lost sight of, but from my enemy and tormentor with a lightness and buoyancy of spirits, elating my imagination to the belief that I was restored to perfect health as if by a miracle.

"I never heard what was the precise duration of the operation but conceive it to have been between twelve and fifteen minutes. With respect to the pain I am persuaded that if it were possible to concentrate what I have often suffered in one night into the same space of time it would have been less endurable. Indeed, this difference between the operation and one night's endurance of the stone was manifest. I have often been most distressingly reduced by the latter, but was not exhausted in the slightest degree by the former; at least my mind was firm throughout, and my body was not sensibly enfeebled. Upon the whole should I be again similarly afflicted, I should not hesitate in again submitting myself to the same mode of relief provided I could place myself in equally capable hands."

It is pleasant to know that this plucky English gentleman speedily recovered. He ceased to keep his bed at the end of the second week; was able to go downstairs at the end of the third; and walked nearly two miles by the end of the month. He rode on horseback, and was in fact perfectly cured at the end of the sixth week from the day of the operation; but the tone of the sphincter was but slowly regained, especially when walking or standing, and he then found it very difficult to retain his urine.

The portrait of Rau is copied from the illustration in Professor J. A. J. Barge's *De Leidsche Anatomie*, Herdacht, 1923, p. 20.

THE INTERVERTEBRAL DISCS.

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The objects of the research, the results of which are given in this paper, were to verify certain debatable points about the structure of the intervertebral discs, and to determine the mode of nutrition, the state of the discs from infancy to old age, and the diseases to which they are subject.

In the anatomy of the discs the following points were doubtful: The presence of a central synovial cavity; the nature of the cells in the nucleus pulposus; the size and position of the nucleus relative to the annulus; the relationship of the cartilaginous plates to the nucleus and annulus; and the presence of nerves, blood-vessels, and lymphatics. The function of the discs was not accurately known. The state of the discs in successive age-periods from birth to old age had not been systematically studied; nor had the pathological affections of the discs, other than destruction secondary to vertebral tuberculosis. There was thus a considerable amount of knowledge lacking concerning a group of structures which constitute a large proportion of the total length of the movable part and the largest ligamentous system of the spine. It was felt that the determination of this knowledge would aid in the interpretation of the clinical features of spinal affections.

The material for the anatomical and pathological investigation consisted of post-mortem specimens from 57 persons who died in a London Poor-Law hospital. The ages at death ranged from 1 year to 86 years. There were 2 subjects in the first decade of life, 4 in the second, 4 in the third, 6 in the fourth, 13 in the fifth, 10 in the sixth, 10 in the seventh, 7 in the eighth, and 1 in the ninth: 37 were males and 20 were females. Death had been due to a variety of acute and chronic diseases. No account of spinal symptoms was obtainable from the clinical records. One mid-thoracic and one lumbar disc were taken from each subject, permission to remove more not being obtainable.

In addition, with the assistance of Dr. W. H. Coldwell, the files of the X-ray Department of the Royal National Orthopædic Hospital for the years 1928–9 were searched for skiagrams of cases with spinal affections other than tuberculosis. From these and from private records a series of skiagrams showing affections of the discs was obtained.

OBSERVATIONS.

Anatomical.—The discs taken from each subject were sectioned horizontally and vertically and examined both macroscopically and microscopically. On macroscopic examination were noted the structure, shape, consistency, colour, compressibility, and elasticity. All these differed considerably in

different subjects; the observations made on them are described below under the headings of 'age changes' and 'pathological changes'. In the main, in the healthy discs of young subjects the observations are confirmatory of previous descriptions. It was noted that the size and position of the nucleus pulposus relative to the annulus fibrosus varied considerably; but as only two discs from each subject were examined, it cannot be stated whether or not this variation is according to any plan. In some specimens the nucleus was central, in others nearer the posterior or anterior margin; in some it formed a quarter of a horizontal section through the centre, while in others the proportion was as much as two-thirds. The line of demarcation between nucleus and annulus was sharp in healthy discs from young subjects, but ill-defined in all other discs.

On microscopic examination certain features were observed which have not been previously described. It was noted that the cartilaginous plates which bound a disc above and below are properly parts of the disc rather than

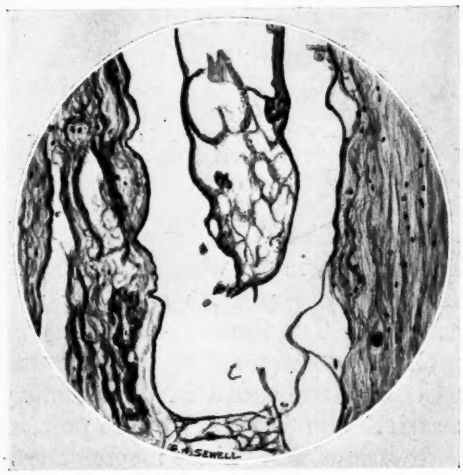

FIG. 212.—Cavity with villus in centre of the disc of a young person. (× 50.)

FIG. 213.—High-power view of wall of cavity. (× 190.)

of the vertebræ, as usually described. They consist of hyaline cartilage that is identical in structure with articular cartilage. One surface of each plate is applied to the cancellous tissue of the corpus vertebræ; on this surface bone-formation was seen to be taking place in the discs of subjects up to 18 years of age. The other surface is broken up into fibrillæ, which radiate into the tissue of the nucleus pulposus; between the fibrillæ, which are composed of hyaline ground substance, cells were seen transitional in character between cartilage cells and fibroblasts. At the margin of the plates there is gradual transition of the hyaline cartilage into the fibrocartilage of the annulus. The presence of the epiphysial centres of ossification in the periphery of the plates was noted in the discs of adolescent subjects.

Previous descriptions of the microscopic appearance of the annulus fibrosus —namely, obliquely running, parallel laminæ of white fibrocartilage deeply, with a few superficial layers of ordinary fibrous tissue—were confirmed. In

the nucleus pulposus the striking feature of a central cavity was observed. In the disc of an infant (*Figs.* 212, 213) the extent of this space was seen to be almost as great as that of the nucleus, but the lumen was almost completely

FIG. 214.—Nutritive channel containing leucocytes and coagulum in nucleus of a young healthy disc. (× 50.)

FIG. 215.—Nutritive channels containing leucocytes in the annulus of a young healthy disc. (× 50.)

filled by villous processes from the wall. In the discs of older subjects the size and form of the cavity varied considerably; in some in which pathological changes were present a cavity was not seen. The tissue surrounding the cavity was composed mainly of a fine reticulum embedded in a gelatinous matrix. Numerous cells were present and were of three kinds: connective-tissue cells, fibroblasts, and cartilage cells. In the discs of young subjects fibroblasts and fibrous tissue were scanty; but at later ages they were plentiful. Cartilage cells existed singly or in groups of two or three and in varying proportions. Fibrillæ of hyaline cartilage were present, particularly peripherally, where they radiated from the cartilaginous plates. The wall of the cavity was lined by a condensed layer, but there was no endothelium or continuous layer of cells as in synovial membrane. The villi were more cellular than the wall of the cavity and were of irregular size and shape.

FIG. 216.—Nutritive channel containing leucocytes, erythrocytes, and coagulum in the cartilaginous plate of a young healthy disc. (× 50.)

Nutritive channels were observed in sections from young healthy discs.

These were narrow spaces in the nucleus pulposus and annulus containing numerous leucocytes. In the nucleus they were seen in the midst of the reticular tissue, having a thin wall in which endothelial cells were not made out (*Fig.* 214). In the annulus they ran longitudinally between the fibres, and no wall was determinable. They could be traced to the periphery of the cartilaginous plates (*Fig.* 215), where they were clearly continuous with channels which pierced the plates from the marrow of the vertebral bodies (*Fig.* 216). Lining the channels through the plates was a single layer of flat elongated cells. Red blood-corpuscles were not seen in the channels in the nucleus and annulus, but were numerous in those through the plates.

Age Changes.—The macroscopic features of the specimens were as follows: In the first decade the disc was seen to be biconvex, highly compressible, and elastic, having a clear white annulus and a colourless, amorphous, gelatinous nucleus, sharply defined from the annulus (*Fig.* 217). The fibres of the annulus were slightly coarser in the lumbar than in the thoracic region. The cartilaginous plates were thin, translucent, and bluish-white. In the second decade the condition of the discs was similar, except that the fibres of the

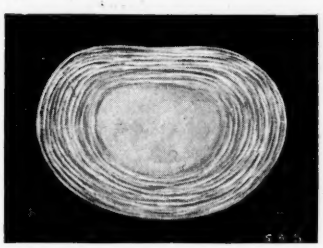

FIG. 217.—Horizontal section of a disc of an infant.

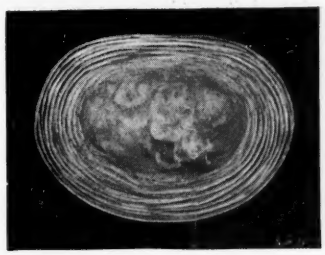

FIG. 218.—Horizontal section of a disc of an adolescent.

annulus were somewhat more coarse and a few fine fibres were present in the periphery of the nucleus (*Fig.* 218). In the third decade the fibres of the annulus were still fine, but the nucleus had become whitish, tougher, and fibrogelatinous, and the line of demarcation between annulus and nucleus less well defined. A few specimens exhibited impaired elasticity, moderate fibrosis, and yellow areas in the nucleus. In the fourth decade most of the specimens had diminished elasticity, moderately coarse fibres in the annulus, and a tough, white, fibrous, and amorphous nucleus. The nucleus passed without clear distinction into the annulus. Pathological appearances were more frequent than in the preceding decade. In the fifth decade pathological changes were common and severe. The fibres of the annulus were coarse, and the nucleus often appeared to be composed of whorls of fibrous tissue (*Fig.* 219). Few discs were more than slightly elastic. With loss of elasticity, the disc was often flattened or even biconcave, and the shape of the surfaces of the vertebral bodies was correspondingly altered. On section of the disc the nucleus did not bulge forward as in infancy, or was even retracted. In the sixth decade further advance was noted in the severity of the changes, the whole disc being inelastic and coarsely fibrous or

fibrocartilaginous (*Fig.* 220). The nucleus was often soft and pultaceous, or dry and friable; in colour it was grey or yellowish. Similar appearances were noted in the discs of subjects between 60 and 70 years. Some of the discs were markedly thinned and partly ossified peripherally; others were fibrous throughout and either not diminished in thickness or even slightly

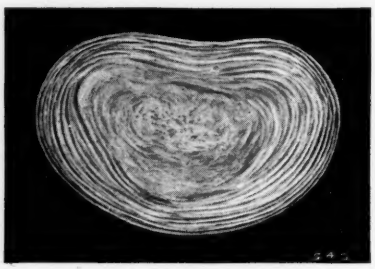

FIG. 219.—Horizontal section of a disc in the fifth decade, showing fibrous transformation.

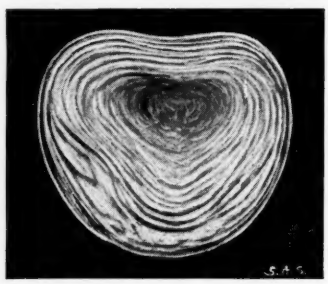

FIG. 220.—Horizontal section of a disc in the sixth decade, showing fibrocartilaginous transformation.

increased. After the age of 70 these changes were accentuated still further (*Fig.* 221).

In the first two decades there were no specimens showing pathological changes, but in the third decade both the thoracic and lumbar discs of one subject showed marked fibrosis with yellow areas. In subsequent decades the incidence of pathological changes progressively increased up to the age of 70, after which it decreased. In each adult age-period except the sixth and seventh, the specimens from one or more subjects showed surprisingly little difference in naked-eye appearance from specimens from young subjects; this was so as late as the eighth decade, in which the thoracic discs from two subjects had semi-gelatinous nuclei and high elasticity, although in one subject the lumbar discs were grossly changed.

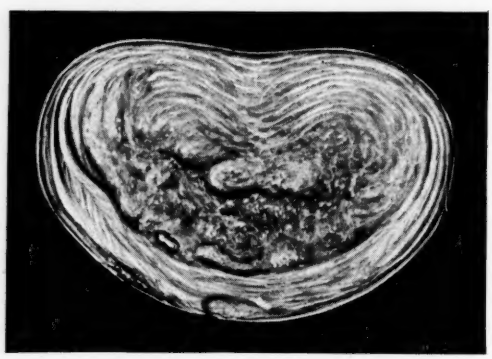

FIG. 221.—Horizontal section of a disc in the eighth decade, showing gross fibrocartilaginous and necrotic changes.

The microscopic appearances observed in the discs of infants were as described above. Sections from discs from older subjects showed cessation of calcification in the cartilaginous plates after the age of 18, gradual increase in the coarseness of the fibres of the annulus and in the proportion of fibrous tissue in the nucleus, increase in the number and size of groups of the cartilage cells both in the nucleus and annulus, and one or more forms of pathological change.

Tables I and *II* show the incidence and degree of changes in the discs in successive decades, and the sex incidence of the changes in the series of the research. The highest incidence is seen to be in the period 50 to 70 years,

with decline in the following decade. The changes were more frequent and severe among the male than the female subjects. They were as common and severe in the thoracic as in the lumbar discs, but the discs in the two regions were not always affected simultaneously or to the same degree; in some subjects the discs in one region were grossly changed, while those in the other region were little if at all altered.

Table I.—Degree of Degeneration of Intervertebral Discs in Successive Decades.

Age	Nil		Slight		Moderate		Severe		Total
	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	
Author's Series.—									
1– 9	2	100·0	0	0·0	0	0·0	0	0·0	2
10–19	4	100·0	0	0·0	0	0·0	0	0·0	4
20–29	2	50·0	1	25·0	1	25·0	0	0·0	4
30–39	1	16·6	2	33·3	2	33·3	1	16·6	6
40–49	1	7·7	6	46·1	3	23·1	3	23·1	13
50–59	0	0·0	0	0·0	3	30·0	7	70·0	10
60–69	0	0·0	1	10·0	2	20·0	7	70·0	10
70–79	1	14·3	1	14·3	2	28·6	3	42·8	7
80–89	0	0·0	0	0·0	0	0·0	1	100·0	1
Norlen's Series.—									
18–30	15	100·0	0	0·0	0	0·0	0	0·0	15
30–40	15	100·0	0	0·0	0	0·0	0	0·0	15
40–50	23	92·0	2	8·0	0	0·0	0	0·0	25
50–60	28	56·0	20	40·0	1	2·0	1	2·0	50
60–70	30	36·6	40	48·8	11	13·4	1	1·2	82
70–80	17	17·0	49	49·0	32	32·0	3	3·0	100
80–89	5	22·7	7	31·8	9	40·9	1	4·6	22

Table II.—Sex Incidence of Degeneration of the Intervertebral Discs.

Degree of Change in the Discs	Male		Female	
	Cases	Per Cent	Cases	Per Cent
Author's Series.—				
Nil	5	13·5	6	30·0
Slight	6	16·1	6	30·0
Moderate ..	9	24·5	5	25·0
Severe	17	45·9	3	15·0
	37	100	20	100
Norlen's Series.—				
Nil	81	48·2	59	36·0
Slight	61	36·3	62	37.8
Moderate ..	18	10·7	43	26·2
Severe	8	4·8	0	0·0
	168	100	164	100

Pathological Changes.—The changes in the discs from the condition in infancy are classifiable into fibrous, cartilaginous, calcareous, bony, fatty, necrotic, and liquefactive. The proportion of fibrous tissue in the discs and the degree of coarseness of the fibres were noted to increase gradually

in successive decades of life. Cartilage cells were seen in the nuclei of discs of infants, but increased in numbers and size of grouping rapidly in later years. It is difficult to say, therefore, whether the fibrous and cartilaginous transformations of discs observed are physiological or pathological. There were, however, in some discs from subjects as young as 20 years advanced fibrosis and cartilage formation, whereas in some specimens from subjects in old age these changes were only moderately or slightly advanced. The relative amounts of fibrous tissue and cartilage were noted to vary. Some discs showed almost complete fibrous transformation, some about equal development of fibrous and cartilaginous tissue, and others great excess of cartilage over fibrous tissue. Changes were present in all parts of the disc, but chiefly in the nucleus. The original reticulo-gelatinous tissue of the nucleus was seen to persist in parts in some specimens even in the presence of advanced formation of fibrous tissue or cartilage. In the nucleus the fibrous tissue tended to assume a whorled form to naked-eye appearance. The cartilaginous hyperplasia took the form of clumps of several cells or of great masses of cells composing nodules.

FIG. 222. — Showing ossification of the annulus and anterior longitudinal ligament.

Fat deposition was observed in many specimens. Necrosis with liquefaction was also common. Small groups of cartilage cells were often seen atrophied, while large nodules were usually necrotic centrally, and frequently disintegrated and liquefied: ragged cavities containing free cartilage cells and débris were thus formed. Complete bony transformation of a disc was not seen, nor was central calcification; but calcified nodules situated peripherally were seen in several specimens; in one there was ossification of the external layers of the annulus and of the anterior longitudinal ligament (*Fig.* 222).

FIG. 223.—Perforation and destruction of the cartilaginous plate of a disc by vascular connective tissue from the vertebral spongiosa. (× 50.)

The cartilaginous plates were noted to be often affected with the rest of the disc. Atrophy of the cells, which were surrounded by a zone of faintly stained matrix, and thinning of the plate was a common appearance. In some specimens fibrous transformation of the matrix was present. In others the cells were grouped into small clumps or even into large nodules, which extended through the whole thickness of the plate, and were continuous with nodules in the annulus or nucleus. In one grossly changed specimen strands of connective tissue containing arterioles were seen perforating and replacing the plates from the spongiosa of the vertebral body

and extending deeply into the centre of the disc (*Fig.* 223). This specimen was greatly thinned, and was composed of coarse fibrous and cartilaginous tissue, much of which was necrotic. The naked-eye appearance is shown in *Fig.* 221.

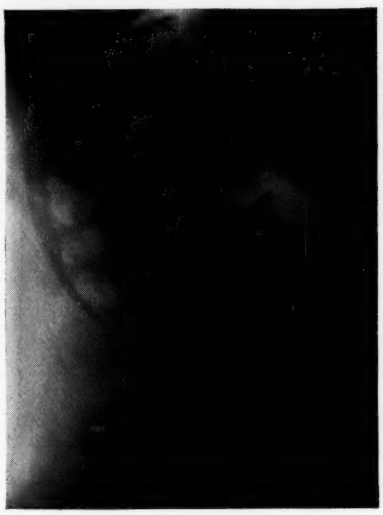

FIG. 224.—Normal appearance of the intervertebral spaces—sharply defined, biconvex, and completely translucent.

FIG. 225.—Thinning and flattening of the discs in a case of severe scoliosis with paraplegia in a young woman.
(*Author's case.*)

FIG. 226.—Peripheral calcification of the discs in a case of thoracic kyphosis in an old man.

FIG. 227.—Central calcification of a disc ('calcinosis intervertebralis') in an old woman: lateral view.

X-ray Appearances.—The following were noted, but no opportunity occurred to correlate them with histological observations: Thinning and flattening (*Fig.* 225); peripheral calcification (*Fig.* 226); central calcification

(*Figs.* 227, 228); obliteration by ossification (*Fig.* 229); diminished transparency with infection of the adjoining vertebræ (*Fig.* 230); thinning, flattening, and diminished transparency with osteochondritis of the adjoining vertebræ (*Figs.* 231, 232); abnormal biconvexity (excessive 'umbilication' of the vertebræ) with rarefying lesions of the vertebral body such as

FIG. 228.—Same case as *Fig.* 227: antero-posterior view.

FIG. 229.—Obliteration by ossification of a disc in a case of healed vertebral tuberculosis.

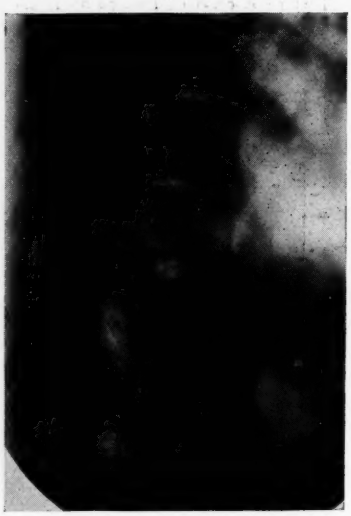

FIG. 230.—Diminished transparency of inflamed discs in typhoid spondylitis.

FIG. 231.—Early appearance of spinal osteochondritis. Thinning, flattening, and diminished transparency of the discs is present as well as fragmentation of the vertebral epiphysial plates.

osteoporosis (*Fig.* 233), carcinoma (*Fig.* 234), and compression fracture (*Fig.* 235). Thinning and flattening were noted in cases of scoliosis and kyphosis, sometimes alone, sometimes in the presence of osteo-arthritis or osteochondritis. Peripheral calcification was seen alone or with osteo-arthritis.

Complete ossification of a disc was observed in only one case—one of healed vertebral tuberculosis at the same site. The significance of the other appearances is discussed below.

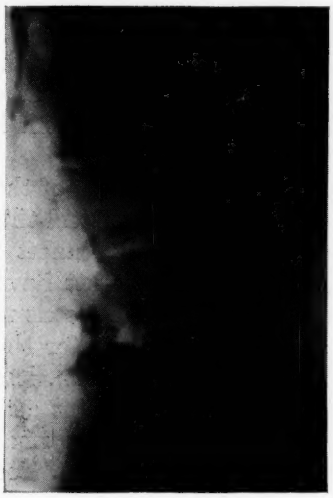

FIG. 232.—Late appearance of spinal osteochondritis. The discs are thinned, flattened, and diminished in transparency, while the vertebral bodies are compressed and irregular.

FIG. 233.—Abnormal biconvexity of healthy discs with osteoporosis of the vertebræ. (*Case of Dr. G. F. Still.*)

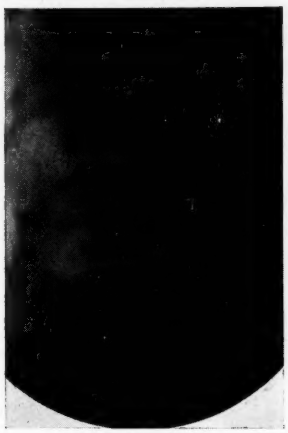

FIG. 234.—Abnormally biconvex healthy discs with carcinoma of a vertebral body.

FIG. 235.—Abnormally biconvex healthy discs with compressed (post fracture) vertebral bodies.

In taking skiagrams of the discs it was found to be essential to take both lateral and antero-posterior views. In lateral exposures the axis of the spine must be parallel with the surface of the film. In judging the depth of the intervertebral spaces the varying direction of projection of the shadow due to the curves of the spine must be taken into consideration.

DISCUSSION.

The subject of the intervertebral discs has been much neglected, particularly by English workers. Apart from short descriptions in anatomical textbooks, there has been no writing on the subject in English since Cleland's paper in 1889.* In other languages there is to be found no writing between Luschka's monograph, published in 1858, and a few papers issued within the last ten years. As these writings are important and not readily available to English readers, it seems a service to include the substance of them in this discussion.

It is well known that the discs form approximately one-fourth of the total length of the movable part of the spinal column, and vary in vertical thickness in the several regions; they are thickest in the lumbar and thinnest in the upper part of the thoracic region. Recently, exact measurements have been made by Jacobi, whose material consisted of the spines of 102 persons ranging in age between 18 and 87 years. He found the smallest values for the discs in the upper thoracic region, where they averaged 3·9 mm., 5 mm. less than those for the lumbar region. Dividing his material into six groups according to age, and into four groups according to the state of the spine—normal, with discs degenerated but no other change, affected with spondylitis deformans, or osteoporotic—he found the proportion of the discs to the vertebræ approximately 1 : 5 in the thoracic and 1 : 3 in the lumbar region. He found further that in his series the proportions remained the same for all age-groups and in each of the four groups according to the state of the spine.

It is also known that the discs in the cervical and lumbar regions are thicker anteriorly than posteriorly and are largely responsible for the forwardly convex curves in those regions; in the thoracic region the discs are thinner anteriorly, in conformity with the posteriorly convex thoracic curve. These truths are important in understanding the effect of pathological changes in the discs upon the shape of the spine.

The discs are usually described as consisting of a peripheral fibrolaminar part (annulus fibrosus) and a central pulpy part (nucleus pulposus). Actually, as I have observed, there are three parts of the disc, distinct histologically and functionally, the third part being the thin plates of hyaline cartilage covering the upper and lower surfaces of the vertebral bodies. These plates are identical in composition with articular cartilage. Luschka measured their thickness and found it to be from 1 to 1·5 mm.

The oblique and cruciate arrangement of the fibres of the annulus is a striking feature, the reason for which has not been quite clear. Labalt observed that the cruciate arrangement is indistinct in the cervical region, more evident in the thoracic, and well-marked in the lumbar. Rouvière has pointed out that the obliquity of the fibres is not the same at all levels of the spine. Measuring the fibres of the anterior part of the discs, he found that in the cervical and thoracic regions the fibres make with the vertical an angle of about 50°, whereas in the lower lumbar region the angle is at least 60°. These measurements are the same in the infant as in the adult. In the horse

* An article by Calvé and Galland, *Journal of Bone and Joint Surgery*, 1930, xii, No. 3, 555, has appeared since this paper was submitted for publication.

the figures for the cervical and lumbar regions are reversed. The peculiar arrangement of the fibres appears to be a provision for taking stresses in the movement of rotation around the vertical axis as well as in the infinite number of directions in which movement can be made around horizontal axes passing through the disc. An explanation, however, is required for the different obliquity of the fibres at different levels of the column, and for the greater obliquity in the lumbar region in man, where rotation is less, than in the cervical region. Rouvière concluded that the inclination on the horizontal of the fibres is proportional to their length, which increases proportionately to the extent of the movements; but that the inclination also increases with the amount of superimposed pressure on the disc. Thus he explained why in the horse the obliquity of the fibres is greater in the mobile cervical region than in the less mobile lumbar region, whilst in man the reverse obtains because of the greater weight the lumbar discs have to bear.

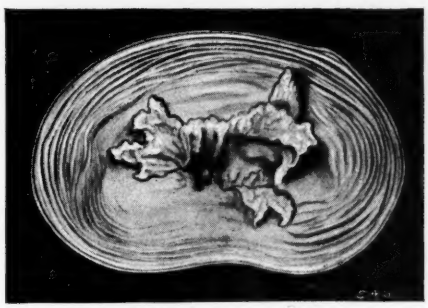

FIG. 236.—Horizontal section (dissected) of an intervertebral disc, showing the synovial-like cavity. (*Luschka.*)

The true nature of the structure of the nucleus pulposus, namely, synovial-like tissue enclosing and infolded into a cavity, was noted by Luschka. He stated that in the infant the cavity is large, occupying more than half the intervertebral space, though filled with villi; in the aged the cavity is commonly narrow, but may be wide (*Figs.* 236, 237). I have been able to confirm these statements by microscopic examination, but not macroscopically. It is clear that an intervertebral disc must be considered, as Luschka stated, a rudimentary diarthrodial joint, in which the cartilaginous plates represent the articular cartilages; the annulus, the fibrous capsule; and the nucleus, the synovial membrane and cavity. In the development of a disc the synovial membrane does not disappear over the articular cartilages, as it does in fully developed diarthrodial joints.

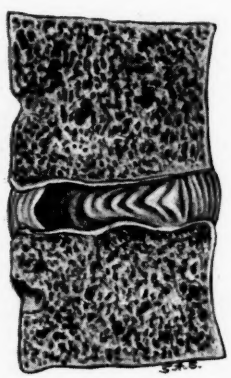

FIG. 237.—Vertical section to show the same feature as *Fig.* 236. (*Luschka.*)

The function of an intervertebral disc does not seem to depend on its arrangement as a joint, but upon the physical difference between its constituents and the formation of the fibres of the annulus. The nucleus pulposus is normally highly compressible and elastic, whereas the annulus is relatively inelastic: a disc thus forms a delicate buffer between the vertebræ, absorbing shocks transmitted through the spine. The fibres of the annulus are strong ligaments between the vertebræ, but by their peculiar arrangement allow a small range of movement in an infinite number of directions. According to Schmorl the position of the nucleus varies in different parts of the spine, so that the weight above is distributed evenly over each disc. I have not been able

to examine a sufficient number of discs in any subject to verify this, though I have noted that the size and position of the nucleus varied considerably in my specimens.

The observation of the presence of nutritive channels in the disc is in conflict with the commonly held view that nourishment is provided by diffusion currents from the spongiosa of the vertebræ. On the other hand, it is confirmatory of the conception of the discs as diathrodial joints. The appearances in my sections establish that nutritive channels exist; they come from the marrow of the vertebral bodies and reach the centre of the disc by first piercing the cartilaginous plates and then running between the fibres of the annulus. I regard them as blood-vessels, but I have not had the opportunity of confirming my observations by injection methods.

Age Changes.—The observations I have made are similar to those of Luschka and Norlen. Luschka described only the appearances of the discs in infancy and old age, and attributed the change to senility. Norlen's investigation was of specimens from 309 subjects between the ages of 18 and 89 years. His observations differ from mine in the following respects: greater frequency of change in females than in males; absence of naked-eye changes before the age of 40 years; and greater incidence and severity of changes in the lumbar than in the thoracic discs (*see Tables I, II*). In both series the nucleus pulposus is clearly the chief seat of change and there is falling-off of the incidence of naked-eye appearances of change in the last as compared with the middle decades of life. The widely varying age of onset and their nature suggest that the changes are not attributable to age alone. Whatever the cause, it is clear that the increasing density and impairment of resiliency of the discs must be responsible for much of the loss of suppleness of the trunk which is usual after the age of 30. Individuals who retain their youthful suppleness after that age do so because they have suffered no changes of their intervertebral discs.

Pathological Changes.—These are divisible into degenerative and inflammatory. Though the fatty, necrotic, and liquefactive changes appear to be degenerative, it is uncertain whether the fibrous, cartilaginous, calcareous, and bony changes are degenerative, inflammatory, or physiological. On the whole, there is gradual increase in the proportion of fibrous and cartilaginous tissue in successive decades of life, and the changes are similar to those of other mesenchymal tissues with advancing years. But fibrous and cartilaginous formations have been seen advanced in subjects as young as 20 years, and have been present in only slight degree in aged subjects. Lyon considers that these changes may occur prematurely through the strain of heavy bodily labour or through the influence of general diseases: in the metabolic disorder alkaptonuria, ochronosis (blackening) of the discs may be present and predispose to degeneration from injury. This is interesting in view of Garrod's observations on the peculiar liability of subjects of alkaptonuria to ochronosis of the articular cartilages of the diarthrodial joints followed by osteo-arthritis, in which, as is remarked below, the pathological appearances are not unlike those seen in the discs. Moreover, Garrod comments on the conspicuousness of the spinal lesions in alkaptonuria. Among other possible causes of the changes may be mentioned contusion and rupture. According to Beneke,

rupture of a disc is by no means rare, resulting in diffuse degeneration at and around the site of rupture. Lyon quotes Schmorl's observation of two cases of laceration of the discs between the 4th and 5th lumbar vertebræ during operations performed in the Trendelenburg position. He also mentions Hoffmann's experiments on the effect of weight-traction on isolated columns of thoracic and lumbar vertebræ, in which it was found that rupture took place always through the lowest disc, the posterior longitudinal ligament being the first point to give way.

Calcification of the centre of the disc has been noted radiologically by many observers since it was originally described by Calvé and Galland in 1922. None of my specimens shows this change, and I have not found it described pathologically by others. In a skiagram a shadow is seen in the centre of one or more intervertebral spaces. This is sometimes single, sometimes partate (*see Figs.* 227, 228). Most often it has been seen in the thoracic and lumbar regions of the spines of persons in middle or later life and has been discovered accidentally. In these cases the appearance seems to have been evidence of degeneration; but in other cases it has been thought to be the result of an inflammation. Baron's case was a child of 12 years; on the eighteenth day of an acute illness with severe pain in the back, lateral curvature with muscular rigidity and increased tendon-reflexes, a skiagram showed a dense transverse shadow between the 12th thoracic and 1st lumbar, and between the 1st and 2nd lumbar vertebræ. These shadows persisted for months, but after a year were no longer demonstrable. The symptoms disappeared after six months with treatment by a corset. Baron assumed that the shadows were due to rapid deposition of calcium in the centre of the discs because of special affinity for calcium of inflamed cartilaginous tissue. Giongo's case was a tuberculous subject, age 44 years, in whom shadows were discovered accidentally in two intervertebral spaces in the thoraco-lumbar region during the course of an examination of the alimentary tract. Giongo thought, therefore, that tuberculosis played a part in the process in the discs. Buffnoir described five cases with slight kyphosis and severe pains in the back radiating to the legs, which ran an intermittent course; dense shadows in the intervertebral spaces were visible in skiagrams. Lyon, on the basis of recent investigations by Schmorl, maintains that in Baron's case the lesion of the disc was secondary to one in the corpus vertebræ; he describes the case as 'spondylitis gripposa with secondary involvement of the discs'. Baron gave the name 'chondritis intervertebralis acuta'. Other writers use the term 'calcinosis intervertebralis' to denote, generally, a calcified area in the disc.

The effect of infectious diseases on the discs has been studied by Schmorl. X-ray examination of vertebral columns affected by infectious diseases, particularly typhoid fever, revealed diminished transparency and thickness, and later, obliteration of the intervertebral spaces. Nearly always the lumbar region was the one affected. At an early stage, when the spongiosa of the vertebræ has become inflamed, the normal elasticity of the as yet unaffected discs exerts pressure which causes collapse of the diseased parts of the adjoining vertebræ. Later, the discs are themselves inflamed by extension from the vertebræ, vascularized, and finally shrink from fibrotic and degenerative

changes, or become ossified from bone-forming tissue which enters with the vessels. A case is described by Rugh in which in place of the discs between the 3rd and 4th lumbar vertebræ dense vascular connective tissue was found passing into the vertebral bodies. Lyon maintains that inflammation of a disc in infectious disease is secondary to an affection of the corpus vertebræ, and probably never primary. According to the stage at which the process is arrested, there remain excessive umbilication of the vertebræ, narrowing or obliteration of the intervertebral space.

RELATIONSHIP OF AFFECTIONS OF THE DISCS TO OTHER DISEASES.

Arteriosclerosis.—The possible relationship between arteriosclerosis and degeneration of the discs was studied by Norlen. He noted that in his series both affections were chiefly found in old subjects, but their relative incidence in a charted record did not run quite parallel (*Fig.* 238). The difference was slight, the curve of both rising continuously as age advanced, until old age, when it became marked. Arteriosclerosis was found in 100 per cent of persons between 80 and 90 years of age, whilst degeneration of the discs was absent in 20 per cent. Norlen thought also that probably the two processes are not concurrent in the earlier decades, there being no constant evidence of simultaneous onset: indeed, arteriosclerosis appeared to begin at an earlier age. In their more severe forms the two states seemed to occur in direct opposition.

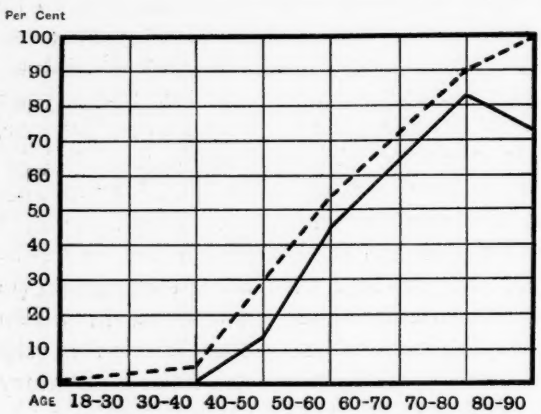

Fig. 238.—Relative incidence of arteriosclerosis and degeneration of the intervertebral discs. The continuous line shows the percentage of cases with degenerated discs in the various decades, and the dotted line the percentage of cases with arteriosclerosis in the same groups. (*Norlen.*)

Osteoporosis.—Norlen also investigated the possible inter-relationship of osteoporosis of the vertebræ and degeneration of the discs. In this, as with arteriosclerosis, his observations were unfinished; but they are sufficiently striking to warrant further investigation. Of 21 subjects showing osteoporosis, he noted that in 8 there were no macroscopic signs of degeneration in the discs, 10 showed only slight changes, and 3 moderately advanced changes. Thus it appeared that osteoporosis of the vertebræ and degeneration of the discs do not arise simultaneously or proceed concurrently. Norlen's impression was that they run in inverse proportion to one another. When the discs degenerate they become flattened or biconcave instead of biconvex as normally. In severe osteoporosis the concavity of the vertebral surfaces is increased, the discs often remaining biconvex (*Fig.* 239). Hence it seems that the etiological factors differ for

the two states, and that some reciprocal adaptability exists between the vertebræ and the discs when either is diseased. Norlen noted the same phenomenon in destructive processes, such as cancer metastasis, in the vertebræ, the discs in the affected region being markedly healthy and elastic. These observations are confirmed by those of Schmorl, who maintains that the healthy discs actually aid by pressure the destructive process in the vertebræ; he observed that spinal curvature with osteoporosis is most severe when the discs are intact.

The radiographic appearance of the condition of healthy discs with osteoporosis of the vertebral bodies is excessive umbilication of the upper and lower surfaces of the vertebræ (*see Fig.* 233). Umbilication is necessarily a normal skiagraphic feature because of the biconvex shape of the discs. Exaggeration of this feature may not be significant of disease, since it may be present with lordosis, when presumably the nuclei of the discs are pressed forward and thus exert abnormal pressure on the vertebral bodies.

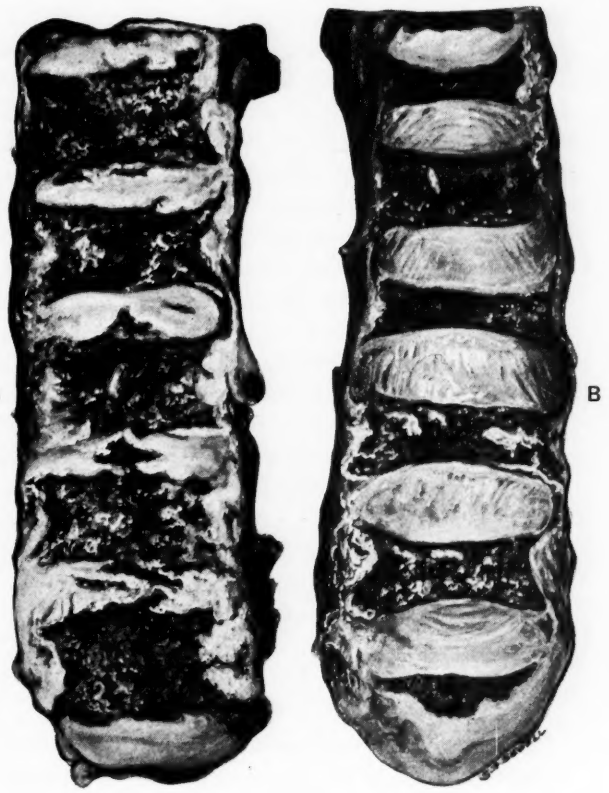

FIG. 239.—Coronal sections of two vertebral columns. A, Degenerated discs with only slightly osteoporotic vertebræ. B, Healthy discs with severely osteoporotic vertebræ. (*Norlen.*)

Osteochondritis of the Spine. — In this condition, which has been recognized recently, the X-ray appearances make it clear that the discs are as much affected as the vertebræ. In cases of recent onset the appearance of diminished thickness and transparency of the discs is as prominent a feature as fragmentation, blurring, and irregularity of the margins of the vertebræ (*see Fig.* 231). In late cases thinning of the discs is as apparent as diminished thickness, antero-posterior wedging, and increased density of the upper and lower margins of the bodies of the vertebræ (*see Fig.* 232). The disease is an affection of the periods of rapid growth in childhood and adolescence, and has its greatest incidence in the thoracic region. Usually several vertebræ and the intervening discs are affected. The symptoms are pain in the affected region and rounded kyphosis or kypho-scoliosis; muscular spasm and local tenderness are present in some cases. The etiology and pathology are conjectural. None of my specimens came from a subject known to have had the disease, and I can find no record of histological examination by others. On

analogy with osteochondritis of other sites it is generally assumed that the affection is inflammatory and infectious.

Osteo- and Rheumatoid Arthritis.—In studying the changes in the intervertebral discs one cannot fail to be struck by the resemblance of these to the changes in joints in chronic rheumatic arthritis. Atrophy and vascularization of the articular cartilages, fibrosis and deposition of fat in the synovial tissue, and thickening and ossification of the capsule are characteristic of the rheumatoid type of chronic arthritis, and are the appearances in many specimens of intervertebral discs. In other specimens formation of cartilaginous nodules, which may calcify, centrally and peripherally, as well as fibrosis of the nucleus and annulus, give a picture similar to that of the osteo-arthritic type. Moreover, the age of maximum incidence of degeneration of discs is the same as of chronic arthritis, and the affections are often present simultaneously and affect chiefly the same regions of the spine. Indeed, assuming, as is believed by many, that traumatism is a causative factor in both, degeneration of the discs can scarcely be present to any degree without leading to chronic arthritis of the spine, and vice versa. When the discs degenerate and lose their elasticity, the bones and joints of the spine are abnormally subjected to the strain of the body weight and to shocks transmitted through them undiminished by the buffer action of the discs. On the other hand, when the spine is limited in movement or deformed by osteo-arthritis or other cause, the intervertebral discs probably suffer minor injuries which result in fibrosis or other change. Manifestly, in a proportion of cases, pain and stiffness of the back attributed to rheumatic affections of the muscles, bones, or joints of the spine are due partly or solely to changes in the discs; also to no small extent deformity from rheumatic arthritis—as well as scoliosis and other chronic affections of the spine—is often due to alteration in the shape and elasticity of the discs.

CONCLUSIONS.

1. The intervertebral discs are rudimentary diarthrodial joints possessing a cavity filled with villi, a fibrocartilaginous capsule, and upper and lower cartilaginous plates comparable to articular cartilages.

2. The mode of nutrition of the discs is by blood-channels which pierce the cartilaginous plates from the marrow of the vertebral bodies.

3. The discs allow a small degree of movement in all directions between the vertebræ, and thus are highly important in imparting flexibility to the spine. They are also powerful buffers between the vertebræ, absorbing shocks transmitted along the spine.

4. In the first two decades of life an intervertebral disc is biconvex and highly elastic; the fibres of the annulus are fine and white, the cartilaginous plates thin, translucent and bluish-white, and the nucleus amorphous, gelatinous, colourless, and sharply defined. The nucleus varies in size and position relative to the annulus. In subsequent decades the disc tends to become progressively more coarse, inelastic, tough, and discoloured. In some individuals the discs preserve their juvenile state until late in life.

5. The lesions of the discs which have up till now been described are

fibrous, cartilaginous, calcareous, bony, fatty, necrotic, and liquefactive changes. A special form which has been noted radiologically is calcification of the centre of the disc, to which the term 'calcinosis intervertebralis' has been applied. Inflammatory lesions have been observed in influenza and typhoid fever. Inflammatory lesions are thought to be secondary to an affection of the spongiosa of the corpus vertebræ. In osteochondritis of the spine the discs are affected as well as the vertebræ.

6. A relationship appears to exist between affections of the disc and other diseases—notably arteriosclerosis, osteoporosis, osteochondritis, and chronic rheumatic arthritis. There seems also to be some reciprocal adaptability between the discs and the vertebræ when either are diseased.

7. (*a*) The usual progressive loss of suppleness of the body after the age of 30 years is due in great part to the increased density and diminished resiliency of the intervertebral discs. (*b*) In a proportion of cases back pain and stiffness, usually attributed to rheumatic affections of other tissues of the back, are due to affections of the discs. (*c*) The symptoms in osteo-arthritis and other chronic affections of the spine are often due in part to changes in the discs. (*d*) The discs when healthy accentuate the effects of rarefying lesions of the vertebral bodies.

8. Diagnosis of lesions of the intervertebral discs depends on radiographic examination. A lateral view with the axis of the spine parallel to the surface of the film is essential, as well as an antero-posterior view. Thinning and flattening, diminished transparency, peripheral or central calcification, obliteration by ossification, or abnormal biconvexity may be observed.

I am indebted to Sir Arthur Keith for suggesting the subject of this research and for criticism and advice; also to him and to Mr. Lawford Knaggs for help in the interpretation of histological features. Dr. W. H. Coldwell has aided me greatly in the collection of the skiagrams, most of which were taken by him; two are by Dr. Leiba Buckley. The work was carried out with the aid of a Research Grant from the British Medical Association, to which I wish to express my gratitude.

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THE MECHANISM OF DEEP TENDERNESS IN GASTRIC AND DUODENAL ULCER.

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In any attempt to elucidate the difficult problem of pain due to lesions of the stomach and duodenum it is essential to distinguish quite clearly between spontaneous visceral pain on the one hand, and objective tenderness on the other. The stomach, as is well known from the work of Mackenzie and Lennander, verified since then by many observers, is quite insensitive to the ordinary mechanical, chemical, or thermal stimuli which cause pain when applied to the sensory somatic nerves. This fundamental fact has caused all the difficulty in explaining the mechanism of visceral gastric pain. We take the existence of true visceral gastric pain as proved, in spite of the teaching of Mackenzie, who regarded it as referred to the nerves of the abdominal parietes. With the question of the precise stimulus adequate to produce it we are not concerned in this paper. Whether it is due to increased intragastric tension (Hurst), to stimulation by hydrochloric acid (Palmer), to vascular congestion in the region of the ulcer (Kinsella), or to some other stimulus as yet unrecognized, is a problem entirely apart from the one with which this investigation deals. Spontaneous visceral pain in gastric and duodenal ulcer sometimes occurs without any tenderness on pressure, and tenderness on pressure may be found apart from any spontaneous pain. The visceral pain is felt in the centre of the epigastrium, but is not localized with any precision by the patient, and does not move with changes of position of the stomach, whereas deep tenderness, as we shall see, is very accurately localized.

CONFLICTING THEORIES ON THE MECHANISM OF DEEP TENDERNESS.

Mackenzie regarded both the spontaneous pain and the objective tenderness of gastric and duodenal ulcers as 'referred' by what he called a viscero-sensory reflex, through the afferent splanchnic nerves and the spinal cord to the parietes. This theory of his led him to minimize the distinction between spontaneous pain and tenderness, and to look for both spontaneous pain and tenderness in the same place. He refers to "the site of pain and hyperalgesia"[1] as though they invariably coincided. It also led him to the belief that the "site of pain and tenderness" was usually remote from the position of the ulcer. In support of this view he quotes several cases in which operative or post-mortem findings appeared to support this contention. It must be remembered, however, that when the abdomen is opened at operation, and still more at post-mortem examination, the relation of the

stomach to the abdominal wall is by no means the natural one. The only method of examination free from this fallacy is that of visualizing the ulcer crater by X rays, and when Mackenzie made his observations radiological technique had not advanced sufficiently to give him this aid.

Hurst, in his Goulstonian Lectures on the sensibility of the alimentary canal (1911), while he rejected Mackenzie's views as to visceral pain, accepted that part of Mackenzie's theory which attributed the objective tenderness to a viscero-sensory reflex. He states: "Pressure applied over a gastric ulcer could not possibly cause local pain, as ulcers in the alimentary canal are insensitive to mechanical stimuli. In the large majority of cases the epigastric tenderness is a result of the irritable focus in the spinal cord produced by abnormal impulses reaching it from the stomach."[2] Like Mackenzie, Hurst fully admits that where the disease process in gastric ulcer extends to the parietal peritoneum, both localized tenderness and muscular rigidity are produced; but these authorities evidently regarded this as an exceptional process, and believed that the common manifestations of deep tenderness in gastric and duodenal ulcer are referred through the agency of the afferent splanchnic nerves and spinal cord to the abdominal wall.

Hurst's Later Theory.—In his latest writing on the subject[3] Hurst takes an entirely new view of visceral tenderness. He subdivides the section on tenderness in gastric and duodenal ulcer into: (1) Visceral tenderness; and (2) Reflex tenderness and rigidity. Under the heading of 'visceral tenderness' he describes a localized deep tenderness which corresponds accurately with the position of the ulcer as visualized by X rays, and shifts with the ulcer in different postures. He concludes that this phenomenon, which had not been observed at the time of his Goulstonian Lectures, can only be interpreted as meaning that the subserous layer of the *visceral* peritoneum over an ulcer is sensitive to pressure. Our grounds for dissenting from this conclusion will be set forth later in this paper. In dealing with what he describes as 'reflex tenderness and rigidity', Hurst still accepts the well-known theories of Mackenzie.

THE SCOPE OF THIS INQUIRY.

It appeared to us necessary in the first place to investigate the relationship between localized points of deep tenderness and the actual lesion in a series of cases of gastric and duodenal ulcer. We hoped by so doing to ascertain whether the point of maximum deep tenderness as determined by palpation does in fact correspond with the position of the underlying ulcer, as Hurst and also Kinsella[4] contend, or whether, as Mackenzie held, they do not coincide. In this way, it seemed to us, we should obtain data which would show whether the facts are consistent with Mackenzie's theory of referred tenderness, or with Hurst's later theory of direct visceral tenderness, or with both theories or neither.

METHOD OF INVESTIGATION.

Only patients who had both a demonstrable ulcer and a localized area of deep tenderness were investigated. In each case the site of the point of

deep tenderness was charted in the ordinary clinical examination: (1) In the supine position; (2) In a lateral position, left or right; and (3) When standing erect. As a rule the stomach was empty when this was done. The stomach was then visualized by barium under the fluorescent screen in the erect position, the point of maximum deep tenderness was marked by a metallic ring, and a radiogram taken. The patient was then placed in the supine position with the ring left *in situ*, the point of maximum tenderness found in this position marked by a second ring, and another film exposed. It was not found practicable to take satisfactory radiograms in the lateral positions. The charts accompanying the case reports given below show the points of maximum deep tenderness in the various positions.

CASE REPORTS.

Case 1.—**A. L., female. Gastric ulcer.**

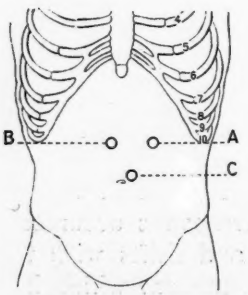

FIG. 240. — *Case* 1. Gastric ulcer. A, Supine; B, Right lateral; C, Erect.

Well localized point of deep tenderness in left of epigastrium in supine position (*Fig.* 240A). When lying on her right side there was a marked shift of the tender point to the right of the mid-line (*Fig.* 240B). In the erect posture the tender point moved downwards 2 in. (*Fig.* 240C).

X RAY.—Erect: ring placed on point of deep tenderness was found to correspond with the visualized crater (*Fig.* 241). Supine: a second ring placed on the new position of the tender point also corresponded with the crater (*Fig.* 242).

OPERATION.—Large ulcer on the lesser curvature and the posterior wall of stomach, with a patch of anæmic tissue on the anterior aspect near the lesser curvature that looked like perforating. Marked induration round ulcer, which was adherent posteriorly. Schoemaker gastrectomy.

FIG. 241.—*Case* 1. Erect position.

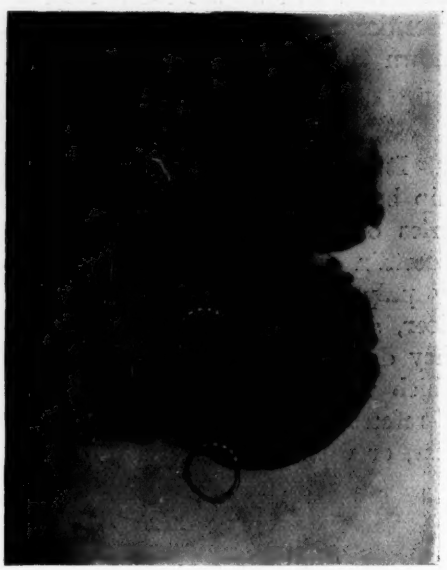

FIG. 242.—*Case* 1. Supine position.

Case 2.—**G. B. S., male. Gastric ulcer.**

This patient had had a perforated ulcer sutured in another hospital seven years before. He now had symptoms of gastric ulcer. Localized point of deep tenderness to left of the mid-line in the epigastrium (*Fig.* 243A). The tender point shifted to the right in the right lateral position (*Fig.* 243B), and downward in the erect posture (*Fig.* 243C).

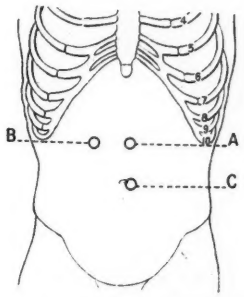

FIG. 243.—*Case* 2. Gastric ulcer.
A, Supine; B, Right lateral; C, Erect.

X RAY.—Erect: the tender point, a little to the left of the umbilicus, corresponded with a visible crater on lesser curvature (*Fig.* 244). Supine: the tender point was higher and again corresponded with the visualized crater (*Fig.* 245).

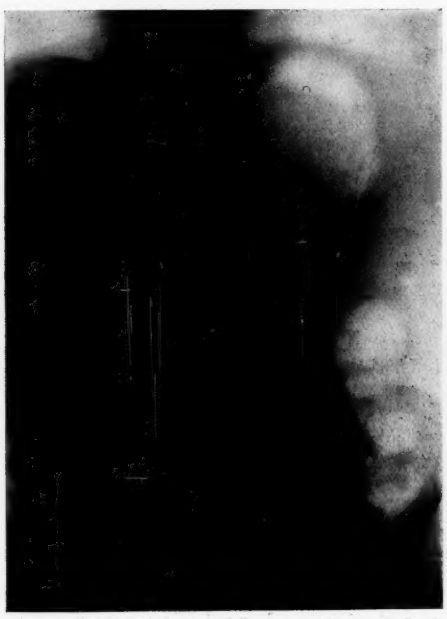

FIG. 244.—*Case* 2. Erect position.

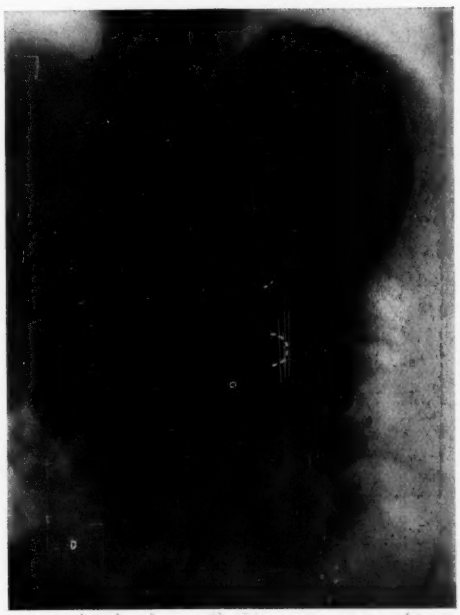

FIG. 245.—*Case* 2. Supine position.

OPERATION.—Small ulcer high up on the lesser curvature, not adherent. Small scar of old ulcer on the anterior wall of first part of the duodenum, now healed. Schoemaker gastrectomy.

Case 3.—**W. L., female. Duodenal ulcer.**

Well localized point of deep tenderness, shifting in the left lateral (*Fig.* 246B) and erect positions (*Fig.* 246C) as compared with the supine (*Fig.* 246A).

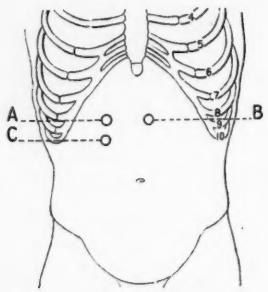

FIG. 246.—*Case* 3. Duodenal ulcer.
A, Supine; B, Left lateral; C, Erect.

X RAY.—Showed that the point of deep tenderness in both erect and supine positions corresponded with the duodenal cap (*Figs.* 247, 248).

FIG. 247.—*Case* 3. Erect position.
U, Umbilicus.

FIG. 248.—*Case* 3. Supine position.
U, Umbilicus.

OPERATION.—Well-marked ulcers on the anterior and posterior walls of the first part of the duodenum. Stippling of serous coat of the anterior wall on handling. No fibrosis over ulcer. Posterior gastro-enterostomy.

Case 4.—J. H., male. **Chronic gastric ulcer.**

No cutaneous hyperalgesia. The deep tenderness was more diffuse than usual (*Fig.* 249), but the difference between its position in the erect and the supine posture was marked. No lateral shift made out when the patient lay on his right side.

X RAY.—The point of deep tenderness corresponded in both positions with the crater on the lesser curvature near the pylorus. Vertical shift of crater and tender point 2 in.

OPERATION.—Large saddle-shaped ulcer on the lesser curvature just above the pylorus, adherent to the liver and pancreas. On the anterior surface of the stomach over the ulcer was a shaggy mass of organized fibrous tissue over an area 2 in. in diameter exactly corresponding to A in *Fig.* 249. Posterior gastro-enterostomy.

Note.—The wide roughened area of stomach wall in contact with the anterior parietal peritoneum should be noted. We believe it to explain the unusually extensive area of localized deep tenderness. The significance of this will be discussed later.

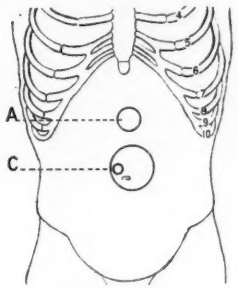

FIG. 249. — *Case* 4. Gastric ulcer. A, Supine; C, Erect.

Case 5.—E. H., male. **Gastric ulcer.**

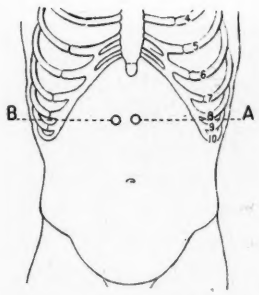

FIG. 250. — *Case* 5. Gastric ulcer. A, Supine; B, Right lateral. No downward shift in erect position.

Point of deep tenderness in centre of the epigastrium (*Fig.* 250A). Slight shift to the right when in the right lateral position (*Fig.* 250B). No downward shift in the erect posture.

X RAY.—The point of deep tenderness corresponded with the ulcer high on the lesser curvature. The point was first marked after a few mouthfuls of barium had been given. When the stomach was completely filled the tender point was 1 in. lower, and in both positions corresponded with the crater. In the supine position the tender point was lower than the visualized crater (? residual tenderness with actual crater now out of reach).

OPERATION. — Small ulcer on lesser curvature. Schoemaker gastrectomy.

Case 6.—L. G., female. **Duodenal ulcer.**

Localized tender point with right lateral and vertical shift (*Fig.* 251).

X RAY.—Supine: There was very well defined tenderness to the right of the lesser curvature, between it and the duodenal cap. Erect: it appeared to be nearer the lesser curvature. It was also found to be lower when the stomach was filled with barium than it had been after the first few mouthfuls. When the patient was again examined in the supine position the tender point was lower than it had been with the stomach empty.

OPERATION.—Well-marked ulcer on the first part of the anterior wall of the duodenum just beyond pyloric ring. No lesion on lesser curvature. A very mobile stomach and duodenum, and the duodenum particularly showed a wide range of mobility. Schoemaker gastrectomy.

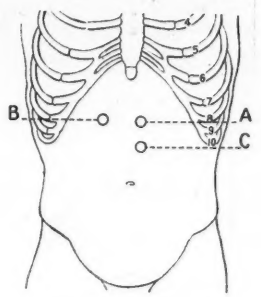

FIG. 251. — *Case* 6. Duodenal ulcer. A, Supine; B, Right lateral; C, Erect.

Case 7.—**A. G., male. Gastric ulcer.**

Localized tender point showing typical shift in the right lateral and erect postures (*Fig.* 252).

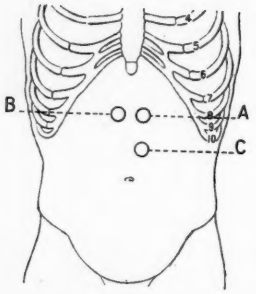

FIG. 252.—*Case* 7. Gastric ulcer.
A, Supine; B, Right lateral; C, Erect.

X RAY.—The tender point corresponded accurately with the crater in both positions (*Figs.* 253, 254).

FIG. 253.—*Case* 7. Erect position.

FIG. 254.—*Case* 7. Supine position.

OPERATION.—Ulcer on the lesser curvature 2 in. above the pyloric ring. Marked fibrosis round it, and petechial stippling on sponging. Schoemaker gastrectomy.

Case 8.—**J. C., male. Chronic duodenal ulcer with pyloric obstruction.**

Tender point to the right of the mid-line, with usual left lateral and vertical shift (*Fig.* 255).

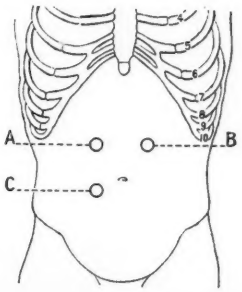

FIG. 255.—*Case* 8. Duodenal ulcer. A, Supine; B, Left lateral; C, Erect.

X RAY.—Stomach full of fluid, and pylorus and duodenal cap not satisfactorily defined. A trace of barium in the cap showed its position. The tender points, with large vertical shift, lay a little to the left of those found before giving barium, and corresponded with the prepyloric segment (*Figs.* 256, 257).

FIG. 256.—*Case* 8. Erect position.

FIG. 257.—*Case* 8. Supine position.

OPERATION.—Duodenal ulcer adherent to falciform ligament by a thick band of fibrous adhesions. Rather extensive roughening of pylorus near this. Stomach very much dilated and hypertrophied and mobile. Posterior gastro-enterostomy.

Case 9.—**L. S., female. Duodenal ulcer.**

Tender point with both right and left lateral shift as compared with supine position. Marked vertical shift (*Fig.* 258).

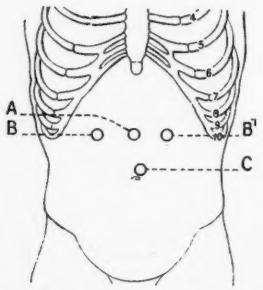

FIG. 258.—*Case* 9. Duodenal ulcer. A, Supine; B, Right lateral; B¹, Left lateral; C, Erect.

X RAY.—Erect: with the stomach filled the tender point corresponded with the duodenal cap (*Fig.* 259). Supine: with the stomach filled the tender point again corresponded with the cap, and now lay well to the right of the mid-line (*Fig.* 260). Evidently the filling of the stomach had carried the duodenal cap further to the right.

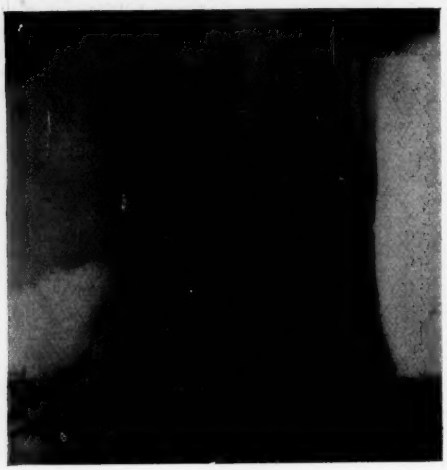

FIG. 259.—*Case* 9. Erect position.

FIG. 260.—*Case* 9. Supine position.

OPERATION.—Duodenal ulcer found immediately under the site of the tender spot, on anterior wall ½ in. beyond the pylorus, with a tuft of organized fibrin over it. Posterior gastro-enterostomy.

Case 10.—A. T., male. Duodenal ulcer.

Tender point with marked vertical shift of 3 in., and slight right lateral shift (*Fig.* 261).

X RAY.—As the stomach filled with barium, the pylorus shifted from a point near the umbilicus, upwards and to the right. The tender point found in the erect position after complete filling was above and to the right of C (*Fig.* 261) and corresponded with the duodenal cap (*Fig.* 262). Supine: the tender point was close to B (*Fig.* 261), and again corresponded with the cap (*Fig.* 263).

OPERATION.—Ulcer on first part of the duodenum, anterior wall, with considerable scarring and stippling, but no adhesions. No gastric ulcer. Posterior gastro-enterostomy.

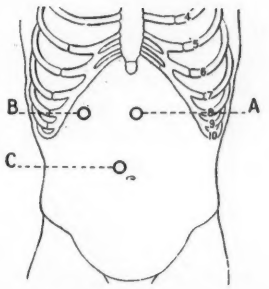

FIG. 261. — *Case* 10. Duodenal ulcer. A, Supine; B, Right lateral; C, Erect.

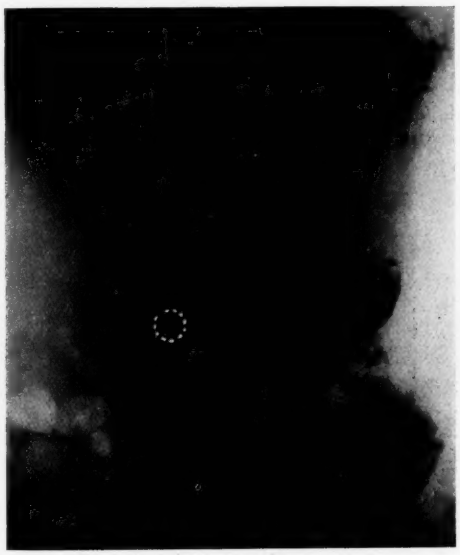

FIG. 262.—*Case* 10. Erect position.

FIG. 263.—*Case* 10. Supine position.

Case 11.—W. E. L., male. Gastric ulcer.

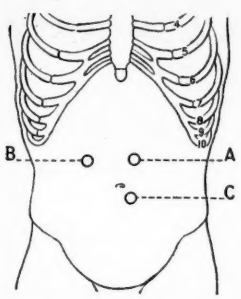

FIG. 264. — *Case* 11. Gastric ulcer. A, Supine; B, Right lateral; C, Erect.

Marked local tenderness. Tenderness in position A (*Fig.* 264), supine, persisted to some extent when the patient was lying on his right side. Also tenderness in position A was still there to a slight extent in the erect posture, though point C (*Fig.* 264) was decidedly more tender.

X RAY.—Erect: after a few mouthfuls of barium deep tenderness at C corresponded with lesser curvature at inner end of a deep persistent incisura. No crater visible. On further filling, the tender point and incisura shifted to the right of the umbilicus. Supine: the tender point was above the umbilicus, and again corresponded with prepyloric position of the lesser curvature.

OPERATION.—Small ulcer on lesser curvature low down. Schoemaker gastrectomy.

Case 12.—**F. B., male. Gastric ulcer.**

Tender point in supine position (*Fig.* 265A) disappeared in the right lateral position. (Probably protected by edge of liver.) Marked downward shift in the erect posture (*Fig.* 265C).

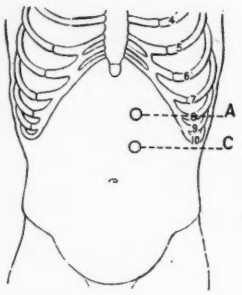

FIG. 265.—*Case* 12. Prepyloric gastric ulcer. A, Supine; C, Erect. Tender point disappeared in the right lateral position.

X RAY.—Deep tenderness corresponded with a prepyloric ulcer crater, and was found very nearly at points A and C in the supine and erect positions (*Figs.* 266, 267). In one film taken with the patient supine the ulcer and ring did not quite correspond owing to the shift of the stomach with expiration. He was then examined to see whether the tenderness moved with the crater in respiration. This was found to be the case, the ulcer and tender point moving 2 in. between full inspiration and expiration.

FIG. 266.—*Case* 12. Erect position.

FIG. 267.—*Case* 12. Supine position.

OPERATION.—Ulcer crater close to the lesser curvature on the posterior wall 2 in. above the pylorus, adherent to the pancreas. On the anterior wall of the stomach at this level, and close to the lesser curvature, was a deposit of organized fibrin. No ulcer in the duodenum. Relation of the ulcer to the liver suggested that our tentative explanation of the loss of the tender point in the right lateral position was correct. The liver margin was lower than usual.

Case 13.—**H. B., male. Duodenal ulcer.**

Definite point of tenderness in mid-line of epigastrium (*Fig.* 268). No lateral shift could be demonstrated (on account of very narrow costal angle ?). No downward shift in erect posture.

X RAY.—The duodenum was high and did not move downwards with standing or with filling. The tender point corresponded pretty closely with it.

No cutaneous hyperalgesia over the tender point, but the patient states that sometimes when spontaneous pain is bad he cannot bear his shirt to touch the spot.

The skin over the tender area was infiltrated with 1 per cent novocain till it was anæsthetic to pin-prick. There was no obvious effect on the deep tenderness.

OPERATION.—Ulcer on first part of the duodenum, anterior wall. Posterior gastro-enterostomy.

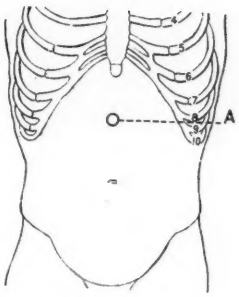

FIG. 268.—*Case* 13. Duodenal ulcer. A, Supine and erect.

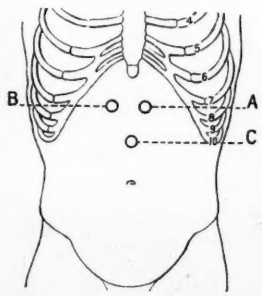

FIG. 269. — *Case* 14. Gastric ulcer. A, Supine; B, Right lateral; C, Erect.

Case 14.—**R. C., female. Gastric ulcer.**

Tender point in supine position to left of the mid-line. Shift to right in right lateral position; 1½ in. vertical shift in erect position (*Fig.* 269).

X RAYS.—With the filled stomach, the tender point, which corresponded accurately with the ulcer crater on the lesser curvature, was ½ in. below that found with the stomach empty. Supine: the tender point and crater again coincided, and were in the same position as A (*Fig.* 269). (*Figs.* 270, 271.)

OPERATION.—Ulcer on the lesser curvature one-third of the way down from the cardiac orifice. A good deal of fibrous roughening over its anterior surface on the stomach wall. Schoemaker gastrectomy.

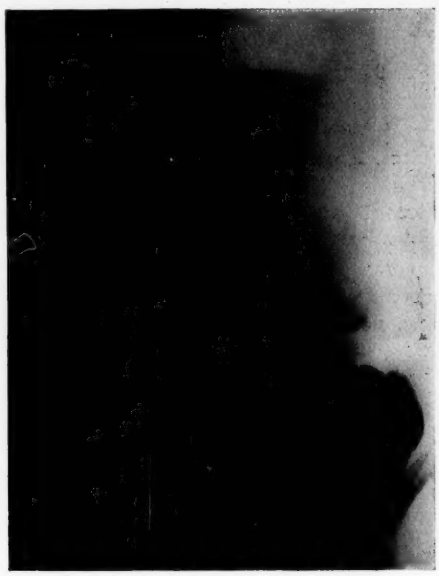

FIG. 270.—*Case* 14. Erect position.

FIG. 271.—*Case* 14. Supine position.

Case 15.—**T. B., male. Duodenal ulcer.**

Well-defined tender point with very slight lateral and vertical shift (*Fig.* 272).

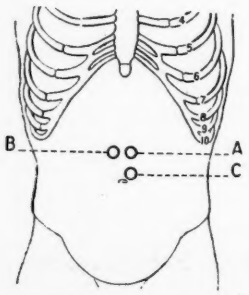

FIG. 272.—*Case* 15. Duodenal ulcer.
A, Supine; B, Right lateral; C, Erect.

X RAY.—Erect: with the filled stomach—the tender point was just to the left of the duodenal cap (*Fig.* 273). Supine: the tender point was slightly higher and again corresponded with the duodenal cap (*Fig.* 274).

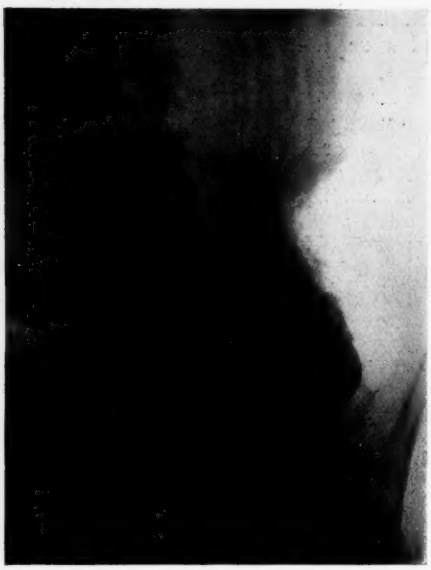

FIG. 273.—*Case* 15. Erect position.

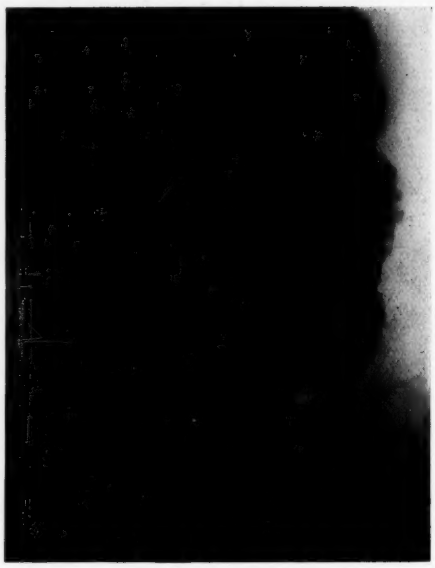

FIG. 274.—*Case* 15. Supine position.

OPERATION.—Ulcer on the first part of the duodenum, anterior wall.

Case 16.—**E. R., male. Gastric ulcer.**

Well-marked tender point. No shift in the right lateral position, but downward shift of 1½ in. when erect (*Fig.* 275).

X RAY.—Filled stomach: the tenderness corresponded in both positions with those previously found, and with the crater (*Fig.* 276, 277).

OPERATION.—Large ulcer high up on the lesser curvature extending to the posterior wall. Duodenum normal. Schoemaker gastrectomy.

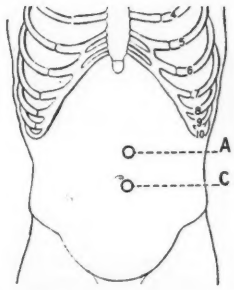

FIG. 275. — *Case* 16. Gastric ulcer. A, Supine and right lateral; C, Erect.

FIG. 276.—*Case* 16. Erect position.

FIG. 277.—*Case* 16. Supine position.

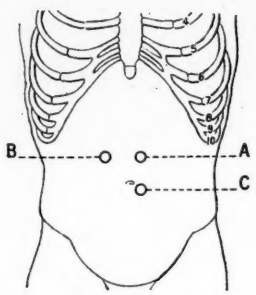

FIG. 278.—*Case* 17. Gastric ulcer. A, Supine; B, Right lateral; C, Erect.

Case 17.—**J. Y., male. Two gastric ulcers.**

A single well-defined tender point supine, with definite right lateral and vertical shift of 1½ in. (*Fig.* 278).

X RAY.—The tender point corresponded with a prepyloric crater in both erect and supine positions. Filling brought it a little lower in the erect posture than that found with the stomach empty.

OPERATION.—One ulcer on the lesser curvature, another on the anterior wall of the stomach 1½ in. above the pylorus.

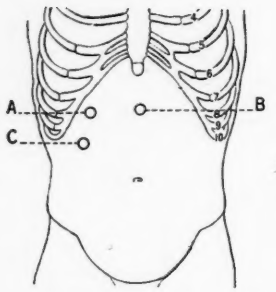

FIG. 279. — *Case* 18. Duodenal ulcer. A, Supine; B, Left lateral; C, Erect.

Case 18.—**E. N., female. Duodenal ulcer.**

Tender point with a slight right lateral and considerable vertical shift (*Fig.* 279).

X RAY.—In both erect and supine positions the tender point coincided with the deformed duodenal cap. The shift with altered posture was 2 in.

OPERATION.—Ulcer on first part of the duodenum. Posterior gastro-enterostomy.

Case 19.—**E. P., female. Duodenal ulcer.**

Tenderness rather more diffuse than usual. Very slight leftward and downward shift with change of posture (*Fig.* 280).

X RAY.—Erect: the tender point coincided closely with the duodenal cap. Supine: the tender point moved upwards, but rather less than the visualized cap. The duodenal cap appeared to be below the liver edge in both positions (*Figs.* 281, 282).

OPERATION. — Small duodenal ulcer, anterior wall, first part. Posterior gastro-enterostomy.

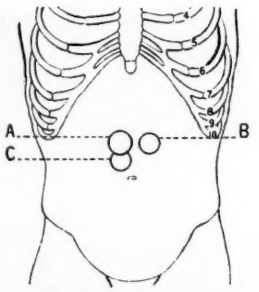

FIG. 280.—*Case* 19. Duodenal ulcer. A, Supine; B, Left lateral; C, Erect.

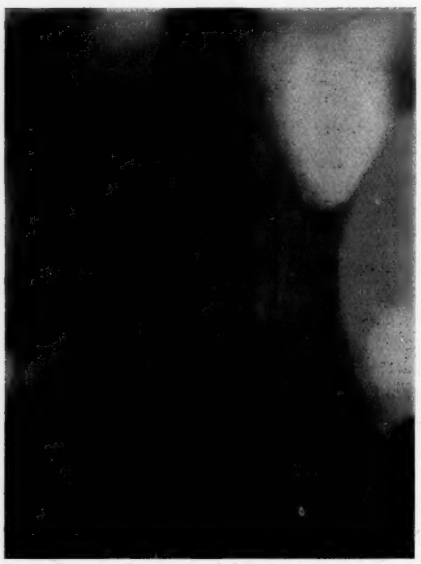

FIG. 281.—*Case* 19. Erect position.

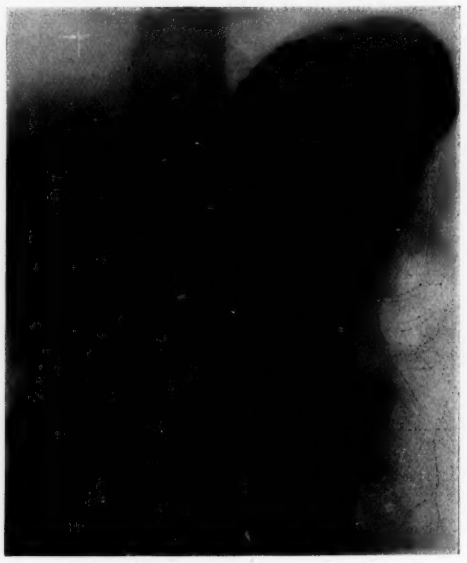

FIG. 282.—*Case* 19. Supine position.

Case 20.—**T. L., male. Duodenal ulcer.**

In the supine position tender point to the right of the epigastrium. Marked left lateral and less marked downward shift (*Fig.* 283).

X RAY.—The point of maximum tenderness accurately corresponded with the deformed duodenal cap in both positions.

OPERATION.—Large duodenal ulcer on the first part of the anterior wall, with some adhesions. Posterior gastro-enterostomy.

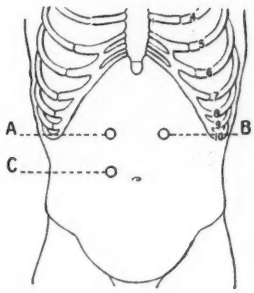

FIG. 283.—*Case* 20. Duodenal ulcer. A, Supine; B, Left lateral; C, Erect.

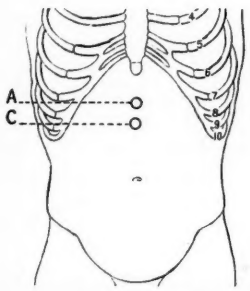

FIG. 284.—*Case* 21. Gastric ulcer. A, Supine; C, Erect. No lateral shift.

Case 21.—**J. L., male. Gastric ulcer.**

Tender point in mid-line of the epigastrium just over a small fatty hernia of the linea alba. (No cutaneous hyperalgesia.) No lateral shift. Slight, but positive, downward shift in the erect position, proving that tenderness was not due to the fatty hernia (*Fig.* 284).

X RAY.—There was about $\frac{3}{4}$ in. vertical shift. The crater was just above the point of deep tenderness in the erect position and a little to the side of it (*Figs.* 285, 286).

OPERATION.—Ulcer on the posterior wall near the lesser curvature half-way up, adherent to the pancreas. Marked thickening and fibrosis of fat on the lesser curvature opposite this, which probably explained why the tender point was to the side of the ulcer. Schoemaker gastrectomy.

Note.—The patient gave no history of pain in the back in spite of adhesions to the posterior parietal peritoneum. The ulcer did not actually invade the pancreas.

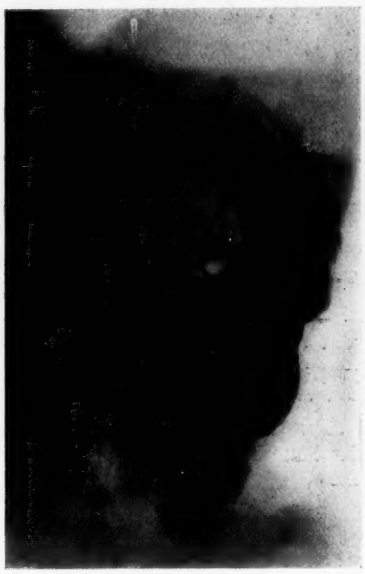

FIG. 285.—*Case* 21. Erect position.

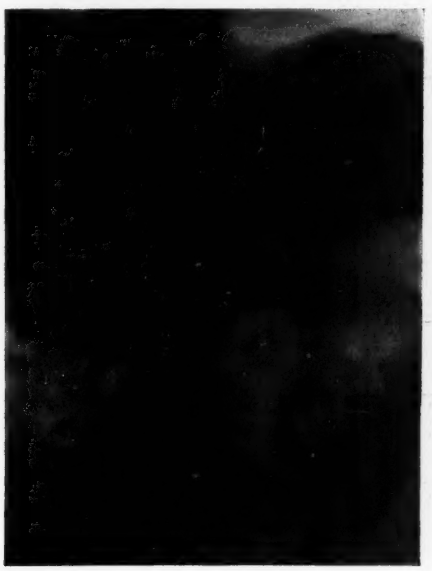

FIG. 286.—*Case* 21. Supine position.

Case 22.—**J. K., male. Duodenal ulcer.**

Usual vertical and right lateral shift of tender point (*Fig.* 287).

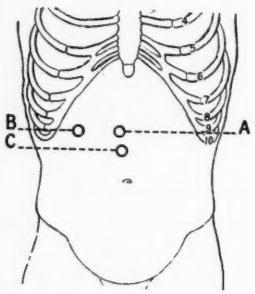

FIG. 287.—*Case* 22. Duodenal ulcer.
A, Supine; B, Right lateral; C, Erect.

X RAY.—Erect: the tenderness was over the deformed duodenal cap (*Fig.* 288). Supine: the tender point was higher, but to the left of the visualized cap, which lay high up under the liver (*Fig.* 289).

FIG. 288.—*Case* 22. Erect position.

FIG. 289.—*Case* 22. Supine position.

OPERATION.—Marked ulcer on the anterior wall of the duodenum, first part. Posterior gastro-enterostomy.

Case 23.—**M. C., female. Duodenal ulcer.**

Tender point with 3 in. vertical shift, but the patient had lost weight, and the abdominal wall and skin were lax. She was also rather confused mentally. In spite of these difficulties, the observations appeared to be sufficiently definite (*Fig.* 290).

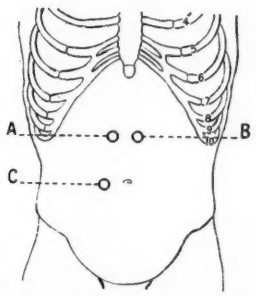

FIG. 290.—*Case* 23. Duodenal ulcer.
A, Supine; B, Left lateral; C, Erect.

X RAY.—The tender point corresponded with the cap in both positions (*Figs.* 291, 292).

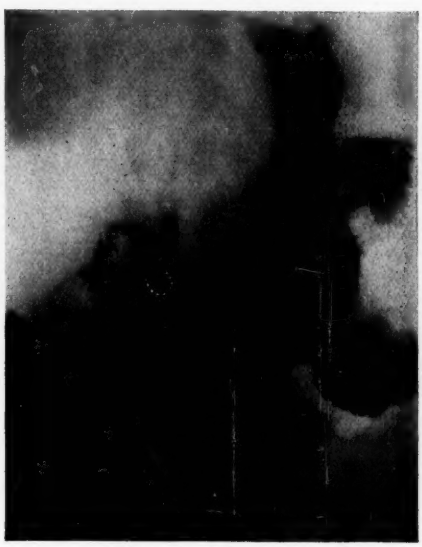

FIG. 291.—*Case* 23. Erect position.

FIG. 292.—*Case* 23. Supine position.

OPERATION.—Ulcer on the first part of the duodenum, anterior wall. Gall-stones. Posterior gastro-enterostomy and cholecystectomy.

Case 24.—**H. W., male. Duodenal ulcer.**

Clinically, definite evidence of shifting of the tender point could not be obtained in the erect posture, because the patient had a trick of tightening up his abdominal muscles and so elevating the stomach on palpation (*Fig.* 293).

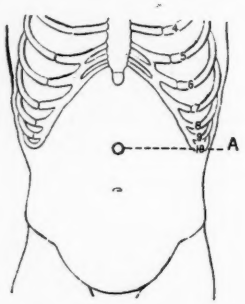

FIG. 293.—*Case* 24. Duodenal ulcer.
A, Supine and erect.

X RAY.—Erect: with a few mouthfuls of barium the tender spot was at the level of the umbilicus, and was just to the left of the duodenal cap (*Fig.* 294). Supine: there was an upward shift of the tenderness of 1½ in., and the point was again just to the left of the visualized duodenal cap (*Fig.* 295).

FIG. 294.—*Case* 24. Erect position.

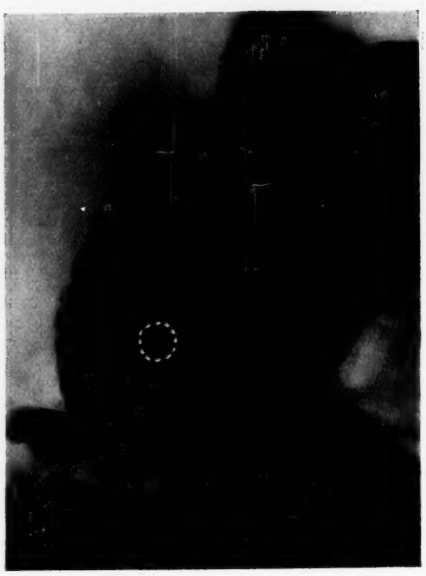

FIG. 295.—*Case* 24. Supine position.

OPERATION.—Large ulcer on the first part of the duodenum, anterior wall. Contact ulcer on posterior wall. Schoemaker gastrectomy.

ANOMALOUS CASES.

Case 25.—**C. R., male. Gastric ulcer combined with appendicitis.**

Point of maximum tenderness in supine position to the right of the umbilicus. No lateral shift. In the erect position the tenderness shifted down 2 in., but there was still some tenderness in the upper point, and also, rather diffuse, in the area between them (*Fig.* 296).

X RAY.—These points did not correspond with a readily visualized prepyloric ulcer crater on the lesser curvature, but point (2) was just below the third (transverse) part of the duodenum. Pressure over the visualized crater elicited deep tenderness in both standing and supine positions, but these points plainly did not correspond with those shown in the chart (*Figs.* 297, 298).

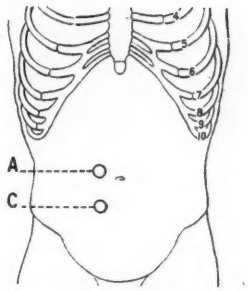

FIG. 296. — *Case* 25. Gastric ulcer and appendicitis. A, Supine; C, Erect.

FIG. 297.—*Case* 25. Erect position. X, Point of maximum deep tenderness found clinically.

FIG. 298.—*Case* 25. Supine position. Arrow points to crater of ulcer.

OPERATION.—Large ulcer on the lesser curvature extending to the posterior wall of the stomach. Duodenum normal. A mass of recent adhesions surrounded the appendix, which was not exposed. Schoemaker gastrectomy.

Comment.—Before operation we considered the case to be very anomalous, but the discovery at operation of evidence of a recent attack of appendicitis accounted for the predominant tenderness in an unusually low position. Deep tenderness over the ulcer was present, but was overshadowed by that due to the peri-appendicular peritonitis.

Case 26.—**M. A. B., female, age 61. Case with shifting tenderness simulating an organic lesion.**

History of pain in the stomach off and on for many years. For the last year or two symptoms have been worse, pain occurring during the night and rousing the patient from sleep. Much flatulence and nausea, but no vomiting. Slight loss of weight and anorexia recently. Pain not related to meals.

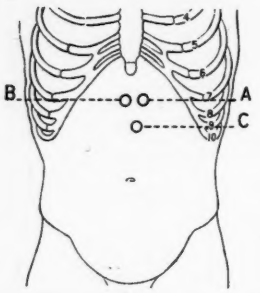

FIG. 299. — *Case* 26. Anomalous case with no organic lesion. A, Supine; B, Right lateral; C, Erect.

ON EXAMINATION.—A well-defined tender point was noted high in the epigastrium to the left of the mid-line (*Fig.* 299), with slight right lateral shift and more marked downward shift in the erect posture.

X RAY.—No ulcer visible, but tender points were marked on the skin, and corresponded with the lesser curvature (*Figs.* 300, 301).

OPERATION.—Stomach, duodenum, and gall-bladder showed no lesion. The appendix was removed, but showed no obvious lesion.

FIG. 300.—*Case* 26. Erect position.

FIG. 301.—*Case* 26. Supine position.

Comment.—This case was one of a small group met with in our investigations in which a typical localized point of shifting deep tenderness was present, without any ulcer or other discoverable organic lesion to account for it. We are unable to explain such cases, but draw attention to them as a caution against the acceptance of a clinically typical point of shifting tenderness as diagnostic of ulcer unless the X-ray appearances confirm the presence of an underlying lesion.

It is as well to point out here that not every visualized ulcer crater is associated with a point of deep tenderness. We have occasionally observed definite ulcer craters, pressure over which elicited no deep tenderness whatever. This absence of tenderness is more common in duodenal than in gastric ulcers, and may be explained by the greater depth of the ulcer from the surface, and the protection afforded to it by the liver.

We may also point out that we have not concerned ourselves with superficial cutaneous hyperalgesia in this investigation. We regard it as so inconstant as to be of no value in diagnosis. In *Case* 13 superficial infiltration of the skin with novocain produced no perceptible diminution of the deep tenderness.

INTERPRETATION OF THE FINDINGS.

We are entirely convinced by these investigations that the correspondence between the tender point and the actual ulcer is real. The coincidence of tender point and ulcer is usually exact, and in most cases in which it cannot be demonstrated on the films as exact, the discrepancy is of a kind which does not exceed the limits of legitimate experimental error. The results obtained on the fluorescent screen were in nearly all cases entirely convincing. We could not in every case reproduce them on films with quite the same precision. Laxity of the skin of the abdominal wall to which the metal rings were attached, varying thicknesses of the abdominal wall, and slight variations in the phase of respiration at the actual moment of taking the film, were all bound to produce slight errors; but the accumulated errors from these causes were in nearly all cases considerably less than the total displacement of both crater and tender point with change of posture. In view of the demonstrable mobility of the first part of the duodenum, notably in spare patients, we were particularly interested, and at first puzzled, to observe what happened when the duodenal cap, deformed by an ulcer, slipped under cover of the liver edge, where it could not be reached directly by the palpating fingers: (1) In some cases the tender point disappeared, particularly when the patient lay on the right side; (2) In others a tender point was found actually to the left of the visualized ulcer now protected by the liver, and usually less easily elicited (*see Fig.* 289). This situation of the tender point to the left of the visualized ulcer, and its diminution in intensity, seemed to us to be explained by partial shielding of the ulcer by the wedge-shaped liver margin from contact with the depressed parietes. These observations differed only in degree from those in cases showing complete disappearance of the tender point, and must have the same explanation.

We are satisfied from these investigations that the correspondence between the point of deep tenderness and the position of the ulcer is a clinical fact which must be accepted and is in most cases readily demonstrable. It should be noted that this is directly in opposition to Mackenzie's observations, which he made before the advent of modern radiological technique.

Our results, therefore, entirely confirm Hurst's recent statements as regards the facts, but our interpretation of them is diametrically opposed to his. Hurst believes that the facts of the relationship between the tender point and the the ulcer must force us to the conclusion that the stomach wall where ulcerated is not insensitive, but is endowed with true visceral tenderness. He believes that the seat of this tenderness is the subserous layer of the *visceral* peritoneum over the ulcer. He has thus abandoned a position which, as we have seen, he accepted in his Goulstonian Lectures in 1911.[2] He has abandoned his earlier belief, not because the body of evidence on which it was based, proving the insensitivity of the exposed viscera to all ordinary painful stimuli, is disproved, but because of these new facts. We hold that there is

nothing irreconcilable between the facts proving the complete insensitivity of the stomach and duodenum, whether ulcerated or not, and the radiological evidence on which Hurst and we ourselves are agreed.

The following case affords direct evidence that the ulcerated stomach is not endowed with visceral tenderness :—

C. H., a very thin man, was admitted to the Manchester Royal Infirmary under the care of one of us (J. M.), with symptoms of pyloric obstruction due to chronic ulcer.

On Examination.—The patient had a somewhat diffuse area of well-marked deep tenderness in the centre and right of the epigastrium.

Operation (March 22, 1930).—The anterior abdominal wall was infiltrated with 1 per cent novocain. Perfect anæsthesia of the abdominal wall was obtained. A mid-line incision above the umbilicus revealed an extensive fibrinous deposit on the anterior wall of the prepyloric portion of the stomach, corresponding with the area of tenderness, and a large prepyloric ulcer on the lesser curvature adherent to the liver. The inflamed wall of the stomach adjacent to the ulcer was grasped with toothed forceps without any traction being exerted. No pain was complained of. The transverse colon was then drawn up out of the wound, when some pain was felt from the drag on the transverse mesocolon. An avascular space in the transverse mesocolon was selected and a hole torn in it with two pairs of dissecting forceps, care being taken to avoid any further drag on the root of the mesocolon. This procedure caused severe pain, each movement in stretching the mesocolon and widening the hole calling forth a sharp exclamation from the patient. Accordingly a general anæsthetic was administered and posterior gastro-enterostomy completed under ether.

This case proved decisively that the inflamed viscus adjacent to the ulcer was insensitive to direct pressure, though it had been acutely tender when pressed upon through the sensitive parietes. The only possible conclusion is that some part of the abdominal wall is responsible for the deep tenderness. The parietal peritoneum is at once the most sensitive layer and the one in immediate contact with the stimulus. We believe that contact between the parietal peritoneum and the inflamed visceral peritoneum over the ulcer provides the adequate stimulus, though whether that stimulus is mechanical and due to roughening, or chemical and due to bacterial toxins, we can advance no opinion. There is good reason to believe that the tenderness is produced by a process of radiation from the sensitive parietal peritoneum to the skin and subcutaneous tissue over it supplied by the same cerebrospinal nerves. The belief that the tenderness is not localized by the sensorium in the parietal peritoneum, but in the more superficial structures, is based on the earlier studies of one of us (J. M.) on shoulder-tip pain.[5,6]

Our more immediate concern, however, is to establish the parietal, as against the visceral, peritoneum as the point of origin of the impulse, and of the cerebrospinal, as against the splanchnic, nerves as its afferent pathway. On these points it appears to us that Hurst has expressed views which lead him to an untenable position. He claims that the only possible interpretation of his radiological findings is a true visceral tenderness. According to his view, the same afferent splanchnic nerve-endings in the region of the ulcer must be stimulated whenever pressure is applied over the ulcer. He therefore claims by implication for the splanchnic nerves an accuracy of spatial localization transcending anything that we know of elsewhere in the

body. For even in somatic sensory nerves accurate discrimination between the different points to which a stimulus may be applied depends upon the stimulation of different sets of nerve-fibres.

How the afferent splanchnic nerves can be supposed to appreciate slight changes in the relationship between an ulcer and the abdominal wall, we are quite unable to see. Yet this power of localization, as we have demonstrated is beyond dispute. It must of necessity involve the stimulation of fresh nerve-endings in each position of the ulcer. These nerve-endings can only be in the parietal peritoneum.

SUMMARY AND CONCLUSIONS.

1. The theories hitherto accepted as to the mechanism of deep tenderness associated with gastric and duodenal ulcers are discussed.

2. An investigation of 25 patients with chronic ulcer associated with localized deep tenderness showed that the area of deep tenderness on the anterior abdominal wall corresponded generally with the ulcer crater, and shifted its position with the ulcer.

3. These observations are quite incompatible with Mackenzie's theory of a viscero-sensory reflex.

4. A case is recorded proving that the stomach, when ulcerated and exposed to direct pressure by an incision under local anæsthesia, is insensitive to pressure.

5. Deep tenderness is explained as due to stimulation of the sensitive parietal peritoneum by contact with the inflamed area of stomach or duodenum at the site of the ulcer, and radiation from the nerves of the parietal peritoneum to the more superficial branches of the same sensory cerebro-spinal nerves.

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[2] HURST, *Goulstonian Lectures on the Sensibility of the Alimentary Canal*, 1911, 65.
[3] HURST and STEWART, *Gastric and Duodenal Ulcer*, 1929, 163.
[4] KINSELLA, V. J., *Med. Jour. of Australia*, 1928, Jan. 21.
[5] MORLEY, J., "The Clinical Significance of Shoulder-tip Pain", *Clinical Jour.*, 1925, liv, 617.
[6] MORLEY, J., "The Significance of the Afferent Impulses from the Skin in the Mechanism of Abdominal Pain", *Lancet*, 1929, ii, 1240.

ACUTE OSTEOMYELITIS OF THE SPINE.

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The occurrence of acute osteomyelitis of the spinal vertebræ following upon some slight lesion is not common, but the difficulty of diagnosis, the severity of the symptoms, and the prolonged convalescence make this disease one of great importance, and form my reason for publishing in some detail the four cases described below. My interest in the condition was aroused by the case of my friend Dr. C. (*Case* 1), and subsequently I have collected details of three others, two of which occurred in the practice of Mr. J. Everidge and the other in that of Mr. G. W. Thomas, of Wakefield. I am much indebted to these two surgeons for the help they have given me, and I am particularly grateful to Dr. C. P. Symonds for his analysis from the neurological aspect of *Case* 1 and for the assistance he so ungrudgingly gave through this very difficult and prolonged illness.

These four cases show certain similarities, and may well be classed in one group. The initial cause in three was the same—a boil—and in the fourth the boy had a subcutaneous abscess in the coccygeal region containing pus, giving a pure culture of the *Staphylococcus aureus*; it is possible that this was a secondary abscess, the original focus not being apparent. A feature common to all was an infection of the urinary tract. One patient lost his kidney from multiple abscess. The boy had his kidney explored, though the reason for suspecting it is not mentioned in the notes, and in the other two cases prostatitis occurred, one going on to abscess.

The invasion of the vertebral bodies took place at very varying intervals after the primary lesion. *Case* 3 had no spinal symptoms till nine months after the lesion was confirmed by X rays, ten months after the beginning of her illness. *Case* 1 had symptoms four weeks, and the lesion was confirmed by X rays four months from the beginning. The times for *Case* 2 are three months and fourteen months, respectively, several earlier X-ray photographs proving negative; *Case* 4 started with lumbar pain, and the X ray showed a lesion two months later, a radiogram taken seven weeks earlier proving negative. An abscess formed in three cases, in *Case* 2 not till four years after the initial lesion. In all the vertebral body was affected, the lumbar in three cases and the cervical in one. It may be said, therefore, that these four cases, in spite of the severity of the onset, were really examples of subacute osteomyelitis, and contrast very definitely with, for instance, a series of 24 cases collected by Makins and Abbott,[1] of which 8 were under their own care and all died, the mortality in the 24 cases amounting to 70 per cent.

The disorder seems to have been studied mostly in France at first, and the list of French authors is a long one, starting from the classic paper by Lannelongue.[2] He had seen only one case of osteomyelitis of the lumbar vertebra. Since then, many cases have been reported and several varieties

are described. Labyerie[3] recognized four varieties: (1) A very acute form, which is a true osteomyelitis; (2) A subacute form, limited in extent, which ends in an abscess; (3) An acute or subacute form, which is rather osteoperiostitis and ends in resorption; (4) A chronic form, which is an ankylosing osteo-arthritis. He considered that the true cause was the introduction of a 'pathological agent' into the general circulation. He refers to boils and minor infections round the finger-nails as a cause, and says the micro-organism is the *Staphylococcus aureus*.

Mathieu[4] considers the acute and subacute forms the most important. He says that the acute form is generally due to a common pyogenic microbe, of which the most frequent is the *Sta. aureus*, while the subacute form generally complicates an infectious disease such as typhoid. He also recognizes three other types: (1) Phlegmonous periostitis; (2) External periostitis giving rise to an abscess between the periosteum and the muscles; and (3) Epiphysial or juxta-epiphysial osteomyelitis. He says that injury causes 25 per cent of the cases, and places a skin lesion, such as a boil, in a secondary position; in this he follows Lannelongue.

Etiology.—The surgeons of the eighteenth century were not greatly concerned with the cause of disease, and in Percivall Pott's account (1779) of the disease of the spine which was eventually called after him, there is no attempt to explain its origin. The tuberculous nature of Pott's disease was suspected by the French surgeons in 1825, and gradually the name 'Pott's disease' became synonymous with tuberculous disease of the spine. Thus tubercle as a cause of acute or subacute disease of the vertebra overshadowed any other possible cause, and in my student days we were taught that there was a 'senile' tuberculosis occurring in late adult life. No real evidence was ever produced that these cases were actually tuberculous, and the question may be asked whether they did not include among them cases of infective osteomyelitis, while the more daring may question the occurrence of senile tuberculosis of the spine altogether.

Many cases have now been recorded of acute osteomyelitis of the vertebra following certain specific infections—for example, typhoid fever, pneumonia, gonorrhœa, and rarely syphilis. In the four cases under review, all due to a staphylococcal blood infection, the determination of the lesion to the bodies of the vertebra is very difficult to explain. Many authors have suggested injury as the exciting cause, and cases have been recorded where there has been a clear history of recent local injury (Fraser and McPherson,[5] J. Ramsay Hunt,[6] Lannelongue,[2] and others); but an injury to the body of a vertebra must be rare and there is no suggestion of an injury in the four cases I report. Infection is due to the *Sta. aureus* in the opinion of all observers, and I have been fortunate in obtaining evidence of a pure culture of the staphylococcus in the blood in all my cases.

Site of Disease.—The bodies or the laminæ of the vertebræ may be affected. If the body is the seat of the disease, it seems to attack those of the lower half of the spine. Mathieu[4] puts the order of frequency as lumbar (53 per cent), then dorsal, cervical, and sacral, and says that the posterior arch is more often affected than the body except in the neck. Labyerie[3] says that there is a remarkable predilection for the lumbar region, and Hahno[7]

reported 41 cases in which the cervical region was affected 7 times, the dorsal 12, the lumbar 17, and the os sacrum 5.

Symptomatology.—The symptoms vary according as the body or lamina is affected, and there are local variations due to the part of the spine concerned. The local manifestations may be obscured by the severity of the general symptoms. The onset may be very severe—rigors, high fever, great prostration, with delirium, sleeplessness, and diarrhœa. A high leucocytosis is the rule. Attention may be drawn to the spine by complaints of agonizing lancinating pains radiating along the affected nerves, or by the rigidity of the muscles guarding the infected area, but there may be no local signs of inflammation unless the laminæ are affected, when diagnostic complications may result by the supervention of signs of spinal meningitis. In very acute cases a fatal toxæmia occurs and death ensues in ten to twenty days from the onset. In less acute cases, among which those here recorded should be included, implication of the spine, or at least direct evidence of it, may be delayed for very variable periods.

The time after the onset of the illness at which backache was complained of was 19 days (*Case* 4), 4 weeks (*Case* 1), 3 months (*Case* 2), and 9 months (*Case* 3); and confirmation by the Röntgen rays was possible in 2 months, 4 months, 14 months, and 10 months, respectively. An abscess occurred in three of the four cases: at 4 months (*Case* 1), 10 months (*Case* 3), and 48 months (*Case* 2) after the onset. It is curious that all cases had an infection of the urinary tract, in *Case* 3 severe enough to lead to nephrectomy. In the early stages it seems that one must rely upon two main symptoms, rigidity of the muscles locally and radiating pains, and that these latter symptoms are not easy to assess, is seen by Dr. Symonds' notes in *Case* 1. In the early stages, as is shown by *Cases* 2 and 4, skiagrams are not helpful; they must be taken repeatedly.

Differential Diagnosis.—The important differentiation is from tuberculous disease of the spine, and this is not difficult as a rule. Infective osteomyelitis of the spine affects adults more often than children, is preceded by some disorder such as a boil (though it must be remembered that a trifling ailment like a boil may be overlooked or forgotten), pneumonia, or typhoid fever, and has a severe onset with high temperature and extreme prostration. The three main symptoms of tuberculous disease are deformity, cold abscess, and paralysis, of which deformity is usually the first sign. In infective osteomyelitis deformity is rare owing to the buttressing of the weak spot by new bone ('becs de parroquet'). This is well seen in the radiograms. Pain, and especially lancinating pain, is characteristic of infective osteomyelitis, and is not relieved by rest as in Pott's disease. Abscess is more frequent in infective osteomyelitis than in Pott's disease, but the spinal cord is not affected except in disease of the posterior arch.

Prognosis.—In the acute variety mortality is very heavy. As mentioned above, Makins and Abbott[1] give a mortality of 70 per cent. Mathieu[4] reports Gridel as giving a mortality of 46 per cent, and he himself considered osteomyelitis of the body of the vertebræ as fatal. One finds in the literature reports of many recoveries, and it is probably necessary to draw a definite distinction between acute and subacute conditions as far as prognosis is concerned, the latter usually running a long course ending in recovery.

CASE REPORTS.

Case 1.—Dr. R. C. (Under the care of Drs. Carpmael and Stewart. Dr. Symonds and Mr. Carson in consultation.)

July 31, 1929.—Two carbuncles opened on left side of neck. They did not seem of importance and soon healed, and the patient left for a holiday at Bexhill, and I (H. W. C.) left also.

Aug. 14.—Dr. Symonds reported: "Felt suddenly seedy. Temperature 103°, headaches, vomiting, and generalized pains."

Aug. 23.—Dr. Symonds reported: "Fever has continued about 103°, with an evening rise and profuse sweats. For the last five or six days the leading symptom has been intense pain in the back of the neck; this is diffuse, spreading down to the right shoulder and up to the head. Swallowing is difficult on account of pain. Three or four days ago a profuse crop of herpes labialis."

On Examination.—The patient was gravely ill and exhausted, but mentally clear, with some physical reserve. Passive movement of head in all directions, free and painless within a very small range. No definite point of tenderness in any cervical spine, but tenderness in the transverse processes beneath the sternomastoid about the 3rd cervical. No tenderness or œdema on posterior wall of pharynx. No glands; nothing in chest, heart, or abdomen. All tendon-jerks present and equal. No defect in the cranial nerves, except that on phonation there was no movement of pharynx. White blood-count, 17,000; urine, trace of albumin; blood culture being done.

Diagnosis.—Staphylococcal septicæmia. ? Arthritis. ? Osteomyelitis in neck —probably the former.

Aug. 27.—Dr. Creed reported: "White blood-count, 17,000; polymorphs, 84 per cent. Urine, a trace of albumin, a few colonies of *Sta. aureus*. Blood culture, *Sta. aureus* in pure culture after forty-one hours in each of eight tubes inoculated."

Sept. 5.—On returning from my holiday, I (H. W. C.) found an urgent message. Acute retention of urine had occurred during the last forty-eight hours. I found some induration in the left neck, which seemed to be subsiding, and a curious alteration in the voice, though nothing was seen on examination of the pharynx. The prostate was acutely tender.

Dr. Symonds saw him, and reported: "After he was last seen the patient had much pain in the left shoulder and burning pain and tingling down the ulnar side of the arm, subsequently paralysis of muscles around the shoulder and a large mass of glands beneath the sternomastoid just above the clavicle. During the past forty-eight hours, retention of urine with much spasm and pain. Has had to be catheterized. Prostate very tender (Carson). Fever still high. General condition pretty fair."

Examination.—The left pupil was smaller than the right, otherwise the cranial nerves were normal. There was weakness and wasting of the left deltoid and spinati, otherwise no motor defect. The supinator- and biceps-jerks were absent on the left and present on the right; the triceps-, knee-, and ankle-jerks were present and equal; the abdominals were all four present and equal; and the plantar responses were flexor. The tongue was moist, the general condition fair, and nothing abnormal was found in the chest or abdomen.

Sept. 6.—Suprapubic drain. Pure staphylococci again found in blood and urine. The bladder was drained for a fortnight, at the end of which time the prostatitis had subsided.

Oct. 9.—Dr. Symonds reported: "Since last seen, pain and weakness in the left shoulder improved, but recurrence of pain developed in the neck, travelling down the right arm with paralysis of right deltoid. Had a suprapubic drainage. Carson thought he has had a prostatic abscess. Subsequently improved, but still has a little irregular temperature and occasional bouts of severe pain in neck and arms. Blood culture now sterile."

Nov. 12.—Dr. Symonds reported: "Temperature running high; blood culture negative; blood-count shows secondary anæmia; white cells, 10,000. Looks

better and stronger, but sallow. Has occasional pain behind the right scapula to about the insertion of the right deltoid, but less severe than before. Weakness and wasting of the right deltoid and biceps; absent right biceps-jerk. I could detect no other physical signs. To consult Ryle about possible value of blood-stream disinfectant."

Nov. 19.—Dr. Symonds reported: "Temperature still up. Tender point in right scapula still present, quite definite. Tender over transverse process of 5th cervical vertebra both sides. Heart, ? soft, to-and-from murmur aortic area."

Nov. 28.—Seen in consultation with Drs. Symonds, Carpmael, and Stewart. Some discomfort in swallowing. Swelling noticed in posterior pharyngeal wall. X-ray photograph (Dr. Worth) shows necrosis and considerable destruction of the 4th cervical vertebra (*Fig.* 302). Decided to try immobilization of cervical spine.

Dec. 1.—Immobilization by plaster jacket.

FIG. 302.—*Case* 1. Showing condition of the 4th cervical vertebra.

Dec. 6.—The pharyngeal swelling is now more right-sided, and is large enough to hide the arytenoids. No swelling of neck or deformity or fixation of cervical spine.

OPERATION (Dec. 14).—Operation at St. Peter's Nursing Home, Leigham Court Road. Dr. Frankis Evans gave anæsthetic; Messrs. W. E. Tanner, Carpmael, and Stewart assisted. A quantity of thick pus was liberated from beneath the anterior common ligament by incision behind the sternomastoid. Pure *Sta. aureus* on culture.

SUBSEQUENT PROGRESS.—The temperature fell to normal very soon, but the dressings were most painful owing to some irritation of the posterior auricular nerve which may have been exposed in the wound. The patient improved very rapidly, sleeping well and putting on weight.

May 1, 1930.—Seen. The sinus has completely closed. There is occasional pain under the right pectoral muscle on certain movements, and a tender spot on top of the right shoulder. Flexion of neck is good, rotation less good. Limitation of movement of right shoulder chiefly due to loss of power in deltoid, but abduction causes radiating pain down the arm. There is a very little lateral deviation to left of cervico-dorsal spine. Is wearing a leather jacket, and is back at work.

At no time has any sequestrum appeared, nor is there any evidence of such in the latest X-ray picture. There is a well-marked spur of new bone ('bec de perroquet').

June 24.—Felt as though some small body was stuck in the back of his throat, felt with his finger, started coughing, and brought up two minute sequestra.

May 13.—Dr. Symonds wrote: "If I might add a note upon the points of medical interest, the situation on Nov. 19 was to my mind that the continued pyrexia could only be explained satisfactorily by supposing one of two conditions to exist—either an infective endocarditis or a hidden suppurative focus. Against infective endocarditis was the absence of any definite bruit.

"It was this which determined me, after I had had a talk with Ryle about him, to have him X-rayed round about the parts where he was still complaining of pain.

Looking back, I feel that my attention at this stage should have been drawn again to the possibility of a central lesion to explain the bilateral involvement of 5th and 6th cervical roots, rather than two separate paravertebral lesions.

"Earlier on I had suspected an osteomyelitis of the vertebræ, but had not been able to conceive such a condition producing bilateral root involvement without at the same time compressing the spinal cord, and of this there was no clinical evidence at any time, once the retention of urine had been explained by you upon a local basis.

In retrospect there is another point to which Ryle first drew my attention in the history of the case, which I think is of considerable clinical importance, and that is the appearance of those enlarged glands on the left side of the neck in the early stages of the illness. Ryle's comment upon this was that one would not expect enlargement of the glands in a case of staphylococcal septicæmia, except by direct spread from a suppurative focus along the lymphatic channels; hence this enlargement, though transitory, pointed to some latent suppurative focus in the neck.

"I might add that at the time of my examination on Nov. 12, although I had come to the conclusion that we were probably dealing with a collection of pus somewhere in the bones of neck or shoulders, I was quite unprepared for the picture revealed by X rays, seeing that active and passive movements of the neck were relatively free and painless and there was no tenderness to pressure upon any of the cervical spines. The tenderness to pressure upon the mid-cervical transverse processes, however, which I first noted in my first examination, was a constant feature throughout."

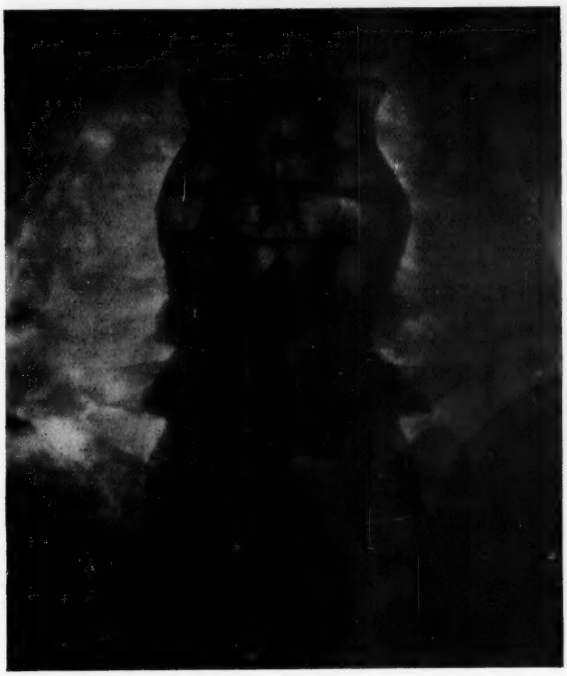

FIG. 303.—*Case* 2. X-ray appearance of 2nd and 3rd lumbar vertebræ.

Case 2.—Dr. S. (Under the care of Mr. Everidge.)

HISTORY.—At the end of 1921, while on holiday convalescing from boils in the neck, the patient was seized with acute retention of urine. He returned to London by ambulance, and was seen by Mr. Everidge, who found he had a prostatic abscess, for which he operated by the perineal route within an hour or two of his arrival in London. Recovery was rapid, but two or three weeks later he developed an acute mastoid, for which Mr. Jenkins operated. He went down to Torquay to convalesce, but while there developed severe pain in the left flank, which became so acute that he had to return to London. No cause for this pain was discovered, nor did the X rays repeatedly taken show anything abnormal. During the succeeding months his pain became worse, and wasting occurred of the left thigh muscles. Finally, X-ray examination showed partial destruction of the bodies of the 2nd and 3rd lumbar vertebræ (*Figs.* 303, 304). He was fitted with a spinal jacket, and his subsequent history is as follows:—

May, 1923.—Superficial abscess over the mastoid region. Healed in ten days.

End of May to middle of August, 1923.—Plaster-of-Paris jacket.

September, 1924.—Herpes zoster.

June, 1925.—Abscess in the right lumbar region extending down into buttock. Operation. Sinus healed by October, re-opened in December, and finally healed in March, 1926. (Ultra-violet rays.)

April, 1926.—Abscess of vertex of the skull after knocking his head on a beam. Large sequestrum removed, exposing the dura.

May, 1927.—Abscess of the right frontal bone followed injury. Sequestrum removed in July.

April-May, 1929.—Pain and swelling in the lumbar renal region. In bed a month. Condition subsided without operation.

Since July, 1929, he has been continuously at work, and in good health apart from backache and some stiffness in the lumbar region. He ascribes his freedom from abscesses since July, 1927, to a course of intravenous injections of perchloride of mercury.

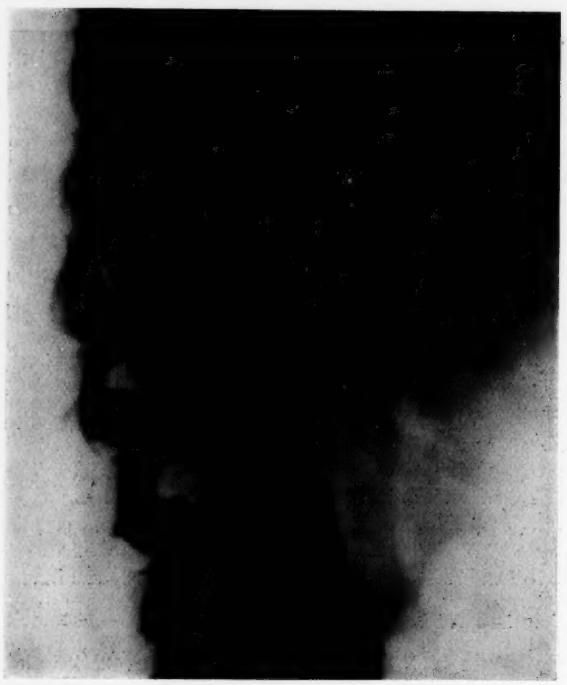

FIG. 304.—*Case* 2. Same as *Fig.* 303: lateral view.

Case 3.—Mrs. B., age 43. (Under the care of Mr. G. W. Thomas.)

Nov. 13, 1927.—What appeared to be simply a small 'boil' on the chin (halfway between the lip and the lower edge of the jaw) was pricked with a sterile needle. No pus found.

Nov. 15.—Redness and infiltration round the 'boil'. Free crucial incision. Fomentations.

Nov. 16.—Extension of swelling, etc., to whole of the chin and lower lip. Free incisions in chin and lip. Condition evidently 'carbuncle' of the chin.

Nov. 18.—Extension to the left side of the mouth and below chin. Further incisions (left side of the chin and just below jaw in the mid-line). Collosol manganese 15 c.c. Antistaphylococcus serum 10 c.c. Wounds treated with H_2O_2, eusol, and flavine.

Nov. 19.—Serum 10 c.c.

Nov. 20.—Collosol manganese 1 c.c.

Nov. 21.—Impaired resonance and weak breath-sounds in the lower half of the right chest posteriorly.

Nov. 22, 24, and 27.—Collosol manganese 1 c.c.

Dec. 1.—Right base explored: 5 oz. of clear fluid (this was not examined). Collosol manganese 1 c.c. Patient still very ill.

Dec. 4.—Collosol argentum 4 c.c. intravenously.

Dec. 5.—Collosol manganese 1 c.c.

Dec. 7.—Collosol argentum 5 c.c. intravenously.

Dec. 11.—Collosol argentum 6 c.c. intravenously.

The patient began to improve about this time. Wounds all healed towards end of December.

Jan., 1928.—Condition not entirely satisfactory, but no definite symptoms.

Jan. 18.—Blood examination showed a marked polymorphonuclear leucocytosis.

Jan. 19.—Right chest explored—negative.

Towards the end of the month some pain and tenderness were noted in the right side of the abdomen.

Feb. 3.—Right kidney enlarged and tender. Urine: Much pus, pure culture *Sta. aureus*, no T.B.

Feb. 20.—Right nephrectomy (lumbar)—some perirenal suppuration. Large tube drain.

May 21.—Urine: Few pus cells, few *B. coli*, profuse culture *Sta. aureus*. (Also on March 30 and April 24.)

June.—Sinus following removal of tube healed about this time.

Sometime after the nephrectomy (about April), the left kidney was a little enlarged and tender, but very fortunately settled down.

July 24.—Urine: Very few pus cells and cocci. The pathologist's report on the kidney said there were numerous pyæmic abscesses in section, and from the pus of one of them a pure culture of *Sta. aureus* was grown.

The patient now seemed well and went away for a holiday, during which she developed backache (lumbar). This gradually became severe and she required veramon tablets, etc., for sleep.

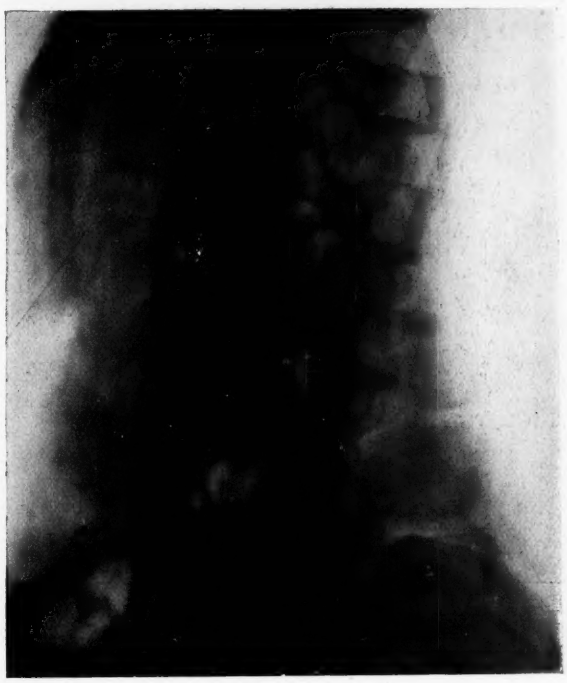

FIG. 305.—*Case* 3. Showing erosion of 2nd lumbar vertebra.

September.—In the middle of this month the nephrectomy wound re-opened and began discharging pus. Examination of the latter showed staphylococci, and culture gave a pure growth of *Sta. aureus*. Guinea-pig inoculated, but when killed showed no signs of T.B. Abdominal and pelvic examination was negative, and apart from the backache there was nothing suggesting spinal trouble. However, as the sinus persisted, an X-ray examination was made and showed "a very distinct lesion of the 2nd lumbar vertebra, which could quite well be an infective osteomyelitis." So I injected the sinus with lipiodol, and this was shown by X rays to pass down a track leading to the 2nd lumbar vertebra and a little way over to the left side. A large probe was also found to pass just to the same spot, the sinus being about 9 in. long. This made the diagnosis quite complete.

December.—The patient was fitted with a Thomas's back support, and allowed to get about, the pain having subsided.

Jan. 30, 1929.—X ray (*Fig.* 305). Traces of lipiodol still present—a little more erosion of the vertebra.

March 3.—A small sequestrum came away, after which the discharge diminished and the sinus was quite healed on March 14. The patient continued to wear the support for some time, and has remained quite well since—free from all symptoms.

June 4.—X ray. "Condition appears to be much improved—no real callus formed, however. Trace of lipiodol still present."

Case 4.—C. B., age 12.

Admitted to King's College Hospital under Mr. Everidge on Sept. 30, 1929, with an eleven days' history of pain in both flanks, chiefly the left, beginning with a rigor. Temperature 104°. Looks ill and flushed, and has circumoral pallor. Poor air-entry over both bases.

Oct. 1, 1929.—Blood-count, 23,200 leucocytes; 82·5 per cent polymorphonuclears.

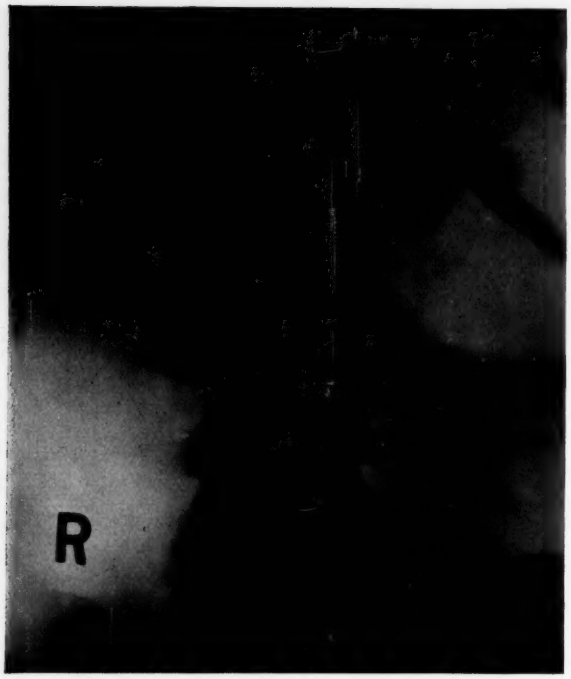

FIG. 306.—*Case* 4. Showing condition of 1st and 2nd lumbar vertebræ.

Oct. 2.—The left kidney was explored, but nothing was found. During the next four days several immuno-blood transfusions were performed. The blood-culture showed staphylococci and enterococci, and the *Sta. albus* was considered to be contaminatory.

Oct. 9.—The blood-culture gave a pure growth of *Sta. aureus*, and the same organism was present in the pus from a subcutaneous abscess in the coccygeal region. Leucocyte count was 45,000.

Oct. 16.—Leucocyte count had fallen to 18,000.

Oct. 31.—Leucocyte count had fallen to 15,000.

Nov. 23.—Some pus was withdrawn from the right elbow-joint, and showed a pure culture of *Sta. aureus*.

Nov. 22.—X-ray examination of the spine showed disease of the 2nd lumbar vertebra and the lower part of the 1st lumbar (*Fig.* 306). On Oct. 1 the spine had been X-rayed and nothing abnormal found except a doubtful narrowing of the joint space between the bodies of D.4 and D.5.

Dec. 1.—He was fitted with a plaster jacket, and sent to Carshalton.

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[1] MAKINS and ABBOTT, *Ann. of Surg.* 1896, xxiii, 510.
[2] LANNELONGUE, *On Acute Osteomyelitis*, Miscellaneous, Pathological and Practical Medicine Tracts 976, 1879.
[3] LABYERIE, *Gaz. des Hôp.*, 1905, lxxviii, 1143.
[4] MATHIEU, *Rev. de Chir.*, 1924, lxii, 96.
[5] FRASER and MCPHERSON, *Lancet*, 1911, ii, 1543.
[6] HUNT, J. RAMSAY, *Med. Record*, 1904, lxv, 64.
[7] HAHNO, *Beitr. z. klin. Chir.*, 1899, xxv, 176.

A CASE OF OSTEITIS FIBROSA

(WITH FORMATION OF HYALINE CARTILAGE).

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Whilst it is hardly possible to add anything useful to the very complete review of this disease by Lawford Knaggs[1] and the brilliant monograph of Dawson and Struthers,[2] the following case seems worthy of record, not only on account of its long history and the very gross changes which appear in the bone, but also because it shows the formation of hyaline cartilage in considerable masses. This formation of hyaline cartilage in osteitis fibrosa is rare, and to this feature I have not been able to find in a search of the already very extensive literature anything more than the scantiest references. Bloodgood,[3] referring to cysts in osteitis fibrosa, says, "in a few of these cases, here and there, islands of cartilage are found". Elmslie[4] states that cartilage occurs in small patches; "it may be hyaline, but is more often fibrous".

CASE REPORT.

History.—The patient was a woman, age 34 years, who came to the Manchester Royal Infirmary in June, 1930, begging for the amputation of her left leg, which she said was useless and painful. She stated that she was well until she suffered a fracture of the left thigh bone twenty years before. This fracture does not appear to have been radiographed, and after treatment it united well. Two years later she noticed a swelling of the shin bone, and found that the limb was becoming shorter than its fellow. This condition grew gradually worse until the left lower limb was short by no less than five inches.

On Examination.—The limb presented a striking picture; the shortening of five inches was found to lie entirely in the femur, the left thigh being stunted, wasted, and marked by deep transverse creases of redundant skin. The tibia was of normal length, but was uniformly enlarged in a fusiform fashion reminiscent of the lower portion of an Indian club. Movements of the knee- and ankle-joints were normal, but the range of the hip-joint was slightly limited in all directions, more especially in abduction. The patient used the limb in walking, but only with difficulty and for short distances; she complained of constant aching pain.

A routine examination showed that the patient was otherwise healthy. No focus of sepsis was to be discovered; the Wassermann reaction was negative. The blood-calcium was normal, and in her thin and very easily palpated neck there was no sign of any thyroid or parathyroid enlargement.

A series of skiagrams of the whole skeleton showed it to be perfectly normal, with the exception of the left lower limb, which gave the remarkable appearance seen in *Fig.* 307. The femur is noticeably shortened and is grossly deformed in contour. The neck is at a right angle to the shaft, and below the neck large irregular bony protuberances are seen to the inner side of the shaft. The normal distinction between cortex and medulla is almost entirely lost, and especially towards the upper end of the bone the appearance is that of numerous, irregular, translucent areas separated by narrow septa of denser bone.

FIG. 307.—X-ray appearance of femur and tibia.

The tibia shows a uniform, fusiform enlargement, greatest at the mid-point of its length. The head and adjacent parts are very translucent; the rest of the bone shows no resemblance to normal structure and has been replaced by an intricate filigree or basket-work of bony trabeculæ outlining translucent spaces. It is noteworthy that the fibula is entirely free from the disease.

OPERATION.—Disarticulation through the hip-joint was done, and recovery was without incident.

PATHOLOGICAL FINDINGS. — The bones were prepared by dissection immediately after amputation. No affection of the soft parts was found, but it was evident that the periosteum was greatly thickened. A section of each bone was made, and the appearance is very accurately depicted in *Figs.* 308, 309. The altered contours of the bones are well seen in section; the cut surfaces are composed, for the most part, of a white fibrous tissue, which was very tough and gritty to the knife. The cut surfaces are marked here and there by small trabeculæ of bone and by points of hæmorrhage, and are flecked by spots of hyaline cartilage, which is seen in considerable masses in the upper third of the tibia. The only cyst disclosed by section of the bones is a small cavity at the mid-point of the femoral shaft, lined by a tough fibous membrane.

A large number of blocks of the tissue were taken, and for these I am indebted to the Pathological Laboratory of the University of Manchester. Out of many examined, those have been selected for illustration which appeared to show the essential features of the disease in this case (*Figs.* 310–317). Evidence of absorption of bone is to be found in most of the microphotographs. It is very difficult to be sure that any of the bone is the original structure, and it is highly improbable that in a

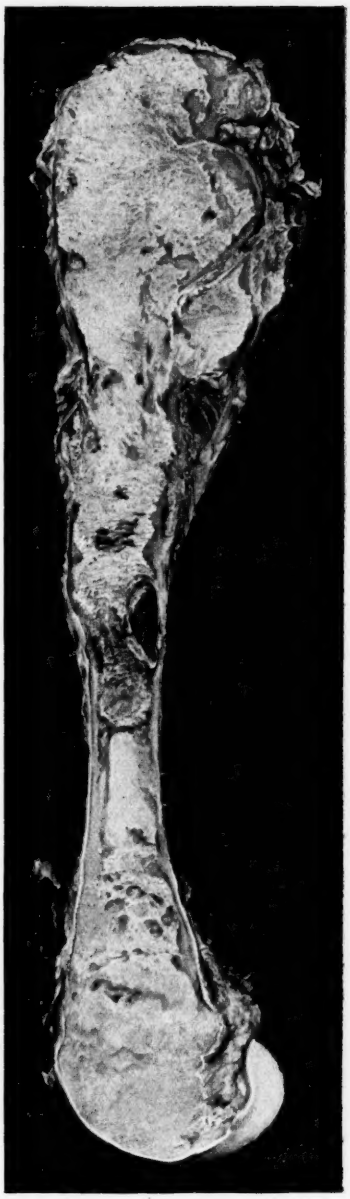

FIG. 308.—Vertical section of the femur. (× ½.)

FIG. 309.—Vertical section of the tibia. (× ½.)

specimen so old and so advanced any portion of the original bone remains. It is never easy to determine the old original bone, and the difficulty is due to the fact that newly-formed bone may, in its turn, be broken down and removed. Dawson,[2] writing of this difficulty, says that he regards as characters of old bone: (1) Haversian systems with definite concentric and intervening parallel

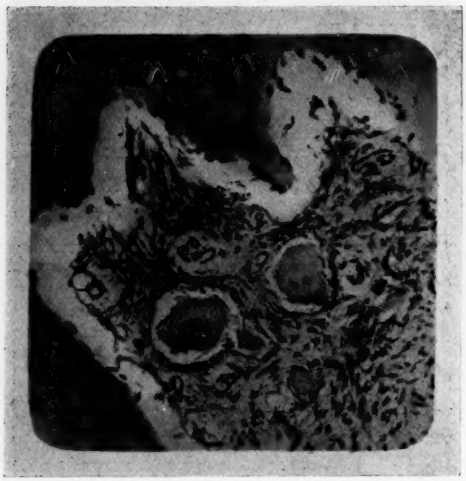

FIG. 310.—The stage of destruction, showing bone being eaten away by osteoclasts. (× 75.)

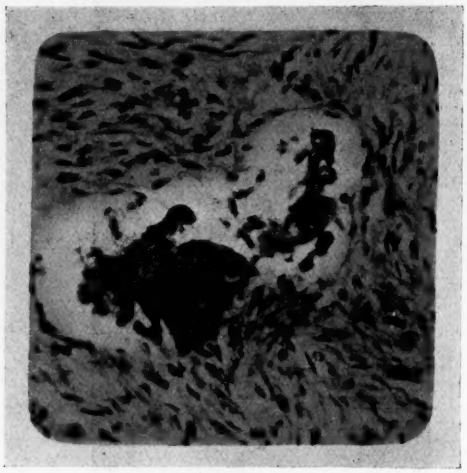

FIG. 311.—A further stage in the destruction. Large osteoclasts surround the almost disintegrated fragments. The early loose and very cellular tissue is shown. (× 150.)

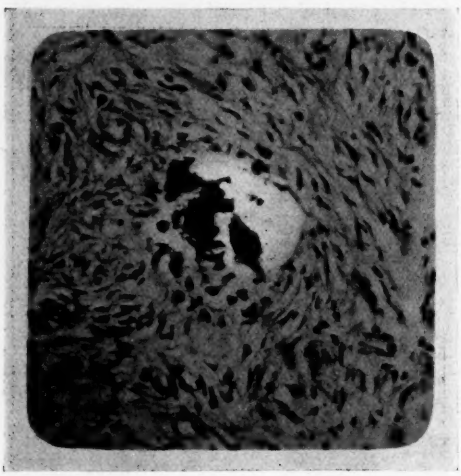

FIG. 312.—A later stage of *Fig.* 311, showing a somewhat denser fibrous tissue. (× 150.)

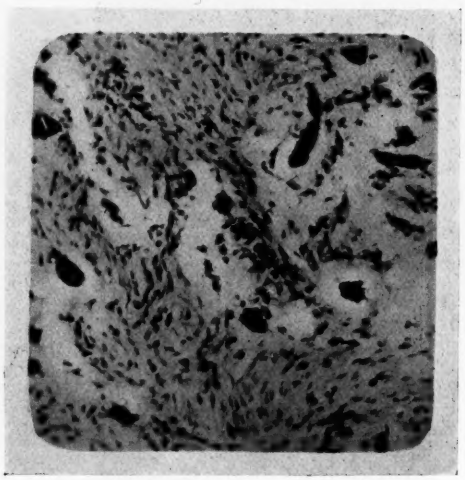

FIG. 313.—The final result of the destruction. Many large syncytial masses lying in a highly cellular fibrous stroma. (× 75.)

lamination; (2) Bone cells uniformly distributed and spindle-shaped, with definite, regular, almost parallel canalicular processes from their sides; (3) Evidences of calcification. On the other hand, he accepted as newly-formed bone that showing: (1) A homogeneous appearance or only slight peripheral

lamination; (2) Cells closely arranged axially and with irregular and radiating processes; (3) Transition at its fibrillated ends into fibrous tissue; and (4) A minimal calcification in the axial portion.

The portion shown in *Fig.* 310 is taken from just beneath the thickened periosteum, and the bone undergoing destruction may possibly be old bone.

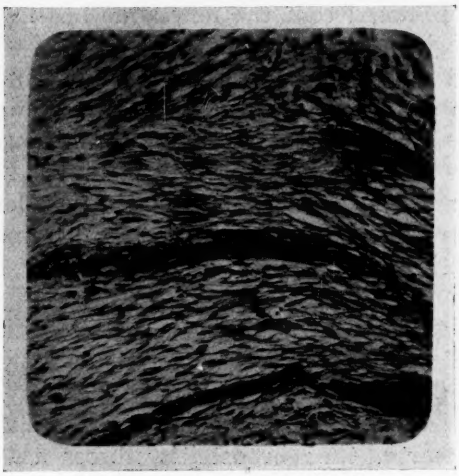

FIG. 314.—Dense, well-formed fibrous tissue showing the beginnings of trabeculæ of new bone. (× 75.)

FIG. 315.—A later stage in the formation of new bone. Many small, mononucleated osteoblasts applied to the growing edge. (× 75).

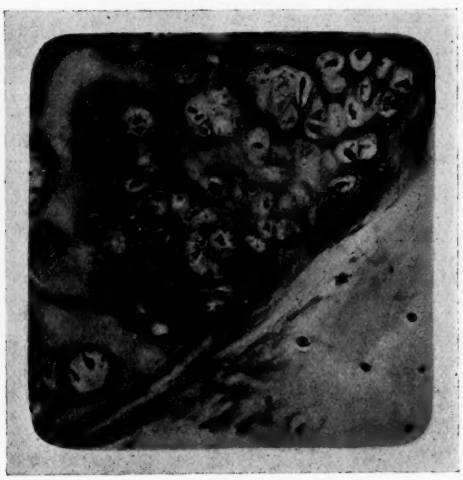

FIG. 316.—A portion of the hyaline cartilage. (× 110.)

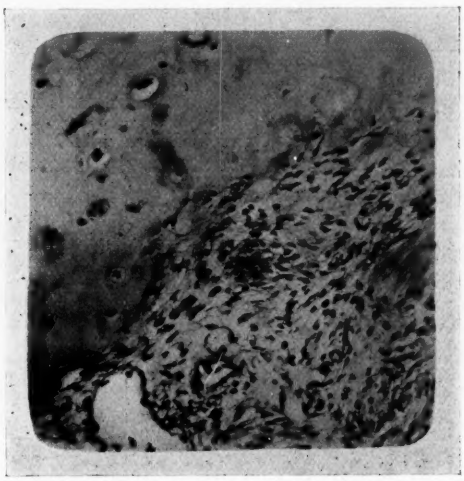

FIG. 317.—The growing edge of the cartilage, showing its origin from the fibrous tissue. (× 110.)

The transition from bone to fibrous tissue is shown in *Figs.* 310–312, until in *Fig.* 313 we reach the condition of a loose fibrous tissue enclosing numbers of large multinucleated giant cells. This tissue, under a high power, suggests great activity of growth, and mitotic figures are common. The resemblance

to an actively growing spindle-celled sarcoma is very close. The ultimate fate of this young connective tissue is the formation of very dense, well-formed fibrous tissue of which the bulk of the altered bone is formed. In many places the formation of trabeculæ of new bone is seen; *Fig.* 314 shows three such areas in which the ground-work of new bone is being laid down in striæ enclosing the future bone cells in its substance. *Fig.* 315 shows a further stage of this process, and it will be noted that the new bone has not the orderly, laminated appearance of original bone.

An unusual feature of the case is the formation of masses of hyaline cartilage. This, as seen in *Fig.* 316, is a true hyaline cartilage, but is crude and imperfect in structure. It is formed by direct growth out of fibrous tissue, and this development is shown in *Fig.* 317. On the right-hand side of the section an area in which chondromucin is being laid down in parallel striæ is well shown. There is nowhere any indication that the hyaline cartilage represents a stage in the formation of new bone.

It is seldom that the opportunity is given to investigate fully an osteitis fibrosa of at least twenty years' standing, and this specimen appears to illustrate all stages of this weird disease. The chief impression left by a study of the sections is that of an intense cellular activity very closely resembling that of sarcoma. It would appear, however, that in osteitis fibrosa this activity is directed to the formation of a dense fibrous tissue and very seldom oversteps the boundary into 'new growth'. In this we have an obvious contrast between osteitis fibrosa and the closely related osteitis deformans, since in Paget's disease the eventual transition into sarcoma is a common terminal event.

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[2] DAWSON and STRUTHERS, "Generalized Osteitis Fibrosa", *Edin. Med. Jour.*, 1923, Oct.
[3] BLOODGOOD, "Bone Cysts", *Ann. of Surg.*, 1910, lii, 148.
[4] ELMSLIE, "Fibrocystic Disease of Bone", *Brit. Jour. Surg.*, 1914, ii, 32.

A CASE OF PRIMARY ADAMANTINOMA OF THE TIBIA.

By ALFREDA H. BAKER,
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CLINICAL NOTES.

(A. H. BAKER)

A MAN, age 46, a locomotive driver, was first seen on Sept. 15, 1930, in the Out-patient Department of Hounslow Hospital. He was complaining of pain in the lower part of the left leg.

HISTORY.—The patient gave a history of having been struck on the left shin by a bar of iron ten months previously. This caused only temporary inconvenience, and there was no wound of the skin. Three months before I saw him he had received another blow on the same place, also from a bar of iron. This injury was very painful, but, again, there was no cutaneous injury. The severe pain lasted for about two hours, and left a dull gnawing pain for a fortnight. Six weeks before he came to hospital he stumbled while carrying a heavy weight on his back, and the jar to his leg caused the pain to return. Local applications of embrocation gave no relief, but he continued working until ten days before his attendance at hospital. He was then seen for the first time by Dr. V. C. Montgomery, who sent him to hospital for an X-ray examination of the leg.

ON EXAMINATION.—A slight swelling of indefinite outline over the lower third of the left leg on its inner and anterior surfaces was observed. The skin appeared to be quite normal; there was no mark of any wound, recent or healed. The swollen area was slightly tender and warmer to the touch than the surrounding skin. Pressure on the sole of the foot caused pain in the leg. Examination of the patient while in hospital produced no evidence of disease apart from the condition of the leg.

The X-ray appearance was as shown in *Figs.* 318, 319. On a tentative diagnosis of myeloma, it was decided to perform a subperiosteal excision and await the pathological report before proceeding to more drastic measures.

Subsequent to the operation the films were shown to Dr. Gordon Thomson, Radiologist to Hounslow Hospital, who was absent from the department during the month of September. He has supplied the following description of the radiological appearance of the leg:—

"There is a tumour which appears as a single lesion in the lower end of the shaft of the tibia. It extends along the medullary canal and has not invaded the epiphysis. The cortex is broken through, reduced in width, and not bulging. In the lateral view, the process of cortical destruction appears to be taking place from outside, rather than from within, the bone. There is some periosteal new bone formation in the tibia, and evidence of periosteal new bone on the outer side of the fibula.

"The X-ray appearance suggests a malignant tumour invading and destroying the bone. There are indications that the tumour may have started on the surface of the bone."

OPERATION (Sept. 17).—Assisted by Mr. Grant Batchelor, I performed a subperiosteal excision of about four inches of the lower end of the tibia. The skin and subcutaneous tissues were normal; the tumour was entirely subperiosteal, and the periosteum was intact but thickened. The bone

FIG. 318.—Antero-posterior view.

FIG. 319.—Lateral view.

was deficient posteriorly over an area of about two inches of the middle part; after removal the lower end broke away. The tumour was soft, greyish in colour, and moderately vascular; it extended downwards almost to the lower epiphysial line, and upwards along the medullary canal. There was considerable sclerosis of the shaft of the bone at the upper limit of the growth, where the medullary canal was almost obliterated.

After the receipt of the pathological report the patient was carefully re-examined for evidence of new growth elsewhere in the body, the skull and

jaws were examined radiologically, and the Wassermann reaction was done. The results of all these investigations were negative.

Convalescence was uneventful. The wound healed by first intention, and the patient left hospital at the end of three weeks with the leg in a plaster-of-Paris splint. Radiograms taken on Nov. 3 show active subperiosteal regeneration of bone. It is proposed to give deep X-ray exposures to the leg with a view to inhibiting local recurrence.

PATHOLOGICAL EXAMINATION.

(L. M. HAWKSLEY)

MACROSCOPIC APPEARANCE OF THE SPECIMEN.—A 4-in. segment of long bone, without periosteum. The upper extremity, which has been chiselled to a blunt point, is intact for $1\frac{1}{4}$ in., but shows no medullary cavity and is of densely hard consistence. A thin shell of compact bone forms half of the circumference of the remaining portion, the other half being largely eroded, with sharply-pointed, undetached fragments of bone extending downwards for 1 to $1\frac{1}{2}$ in., but deficient in the lowest part.

The cancellous portion of all but the extreme upper part of the bone is entirely replaced by slightly lobulated, finely papillary masses of greyish-white, soft, friable tissue, in which on section are seen numerous minute cysts, some containing clear, colourless fluid, others blood. An attempt made to bisect the specimen longitudinally had to be abandoned, owing to the readiness with which the central soft tissue became detached from the bone.

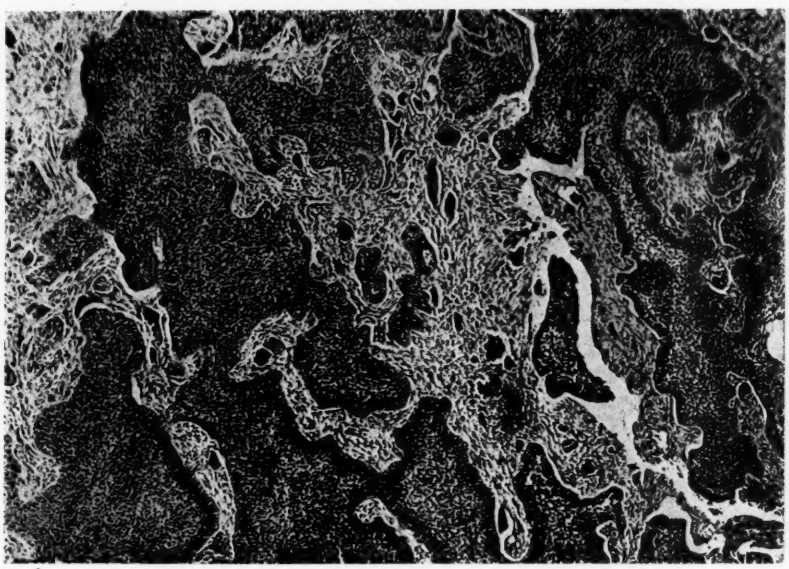

FIG. 320.—Showing general appearance of the epithelial tissue in a fibrous-tissue stroma containing giant cells. (×55.)

MICROSCOPICAL EXAMINATION.—In all the sections prepared from portions of the bone with the tumour tissue on its inner aspect, and also from the soft central growth alone, the histological appearances are similar (*Figs.* 320–322). The outstanding feature is the presence of an infiltrating epithelial

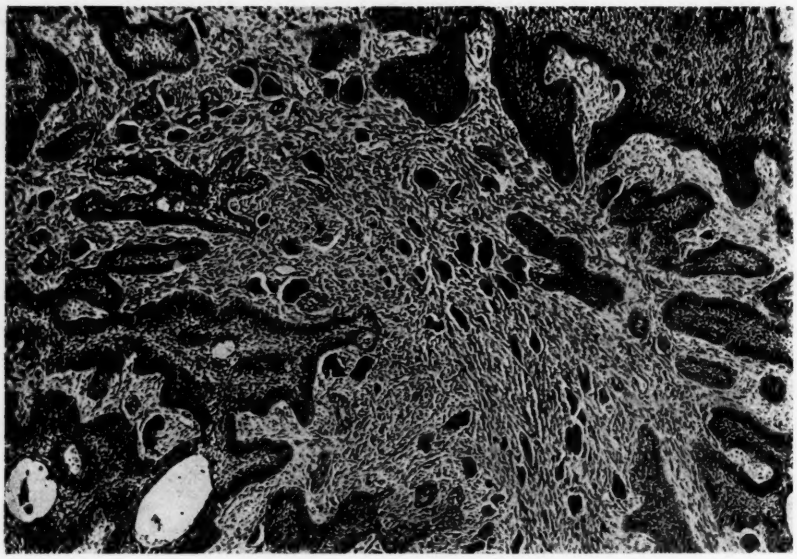

FIG. 321.—Showing multinucleated giant cells in the stroma and cysts in the epithelial network. (×55.)

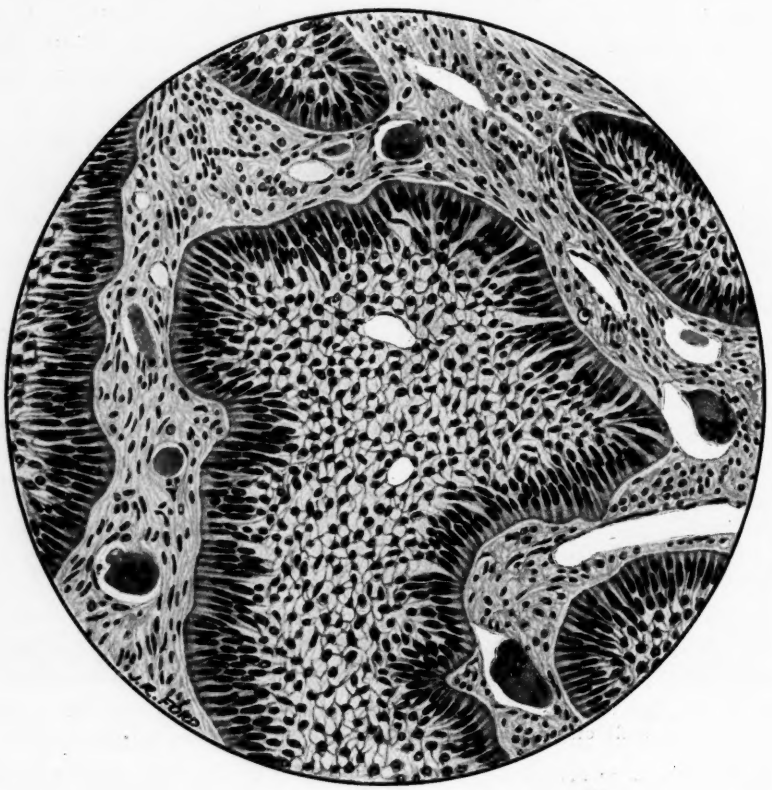

FIG. 322.—Detailed structure of the epithelial tumour, showing stellate cells (representing enamel pulp) with outer layer of columnar cells (representing ameloblasts); two small cystic spaces are seen in the epithelial network. The fibrous-tissue stroma contains multinucleated giant cells, suggesting the epulis type of adamantinoma. (×200).

neoplasm in a fibrous-tissue stroma containing multinucleated giant cells. The appearance of the epithelial tissue at first sight recalls that of rodent ulcer, the growth being formed of masses of small cells arranged in sheets with irregularly projecting processes, and also in narrower strands, with an outer palisade layer of columnar cells; small foci with cystic change simulate gland lumina. The free border of the lobules of new growth is characterized by papillary formation of tissue of the same type.

Epithelial Elements.—

1. *Central cells.*—The centrally-placed cells consist of a network of small spindle-shaped and polyhedral cells with relatively large nuclei and radiating, delicate cytoplasmic processes, which anastomose with those of adjacent cells, the points of communication frequently being marked by a slight thickening. An occasional small whorl of the stellate cells, having a tendency to a more cuboidal form, is also seen. No 'epithelial pearls' or prickle-cells are evident in any of the sections. Spaces of varying sizes, ranging from narrow intercellular clefts to well-formed cysts, are present here and there in the epithelial network; some are devoid of contents, others contain coagulum, while in others there are strands of fibrin with enmeshed blood-corpuscles.

2. *Outermost cells.*—Surrounding each aggregation of anastomosing cells is a single layer of closely-packed, tall columnar cells with terminal bars, which form a continuous, sharply-defined border in contact with the adjacent fibrous-tissue stroma, the less clearly defined ends being directed to the central mass of stellate cells, a layer of cells of intermediate type frequently intervening. The large oval nuclei lie at that part of the cell farthest from the connective tissue; the nucleoli are not conspicuous. An occasional cell undergoing mitotic division is seen.

Stroma.—The stroma consists in the main of fibroblasts, with numerous multinucleated giant cells, whose nuclei number from three or four to thirty or more; in small areas there is more mature fibrous tissue, which has undergone extensive hyaline change. Interstitial hæmorrhage is a prominent feature in some of the sections, and in most of them macrophages containing granules of hæmosiderin are to be found. Isolated spicules of bone are scattered through both the central and the peripheral parts of the masses of tumour tissue. The stroma closely resembles myeloid epulis and benign giant-cell tumour, but there is evidence in many parts that the giant cells function as osteoclasts. Blood-vessels are few, but well-formed. No cysts are found in the stroma, and there is nowhere any indication of sarcomatous change.

Bone.—Sections of decalcified bone from the middle portion of the specimen show only a thin layer of compact substance, its inner aspect encroached upon by the tumour tissue.

Diagnosis.—The epithelial new growth in this specimen has the histological characters of adamantinoma, the stellate cells representing the enamel pulp, and the columnar cells the ameloblasts, of the embryonic enamel organ. In none of the sections is there evidence of enamel formation.

In view of the non-metastasizing properties of adamantinoma of the jaw, and the absence of any history or clinical findings of any other neoplasm in the patient from whom this specimen was removed, the case must be regarded as one of primary adamantinoma of the tibia.

COMMENT.

The tumour is apparently identical with that recognized in the jaw under the names of 'adamantinoma', 'adamantoblastoma', and 'epithelial odontome', the enamel-forming elements of the developing tooth of a four-months' fœtus being represented in their characteristic arrangement. The pseudo-gland formation in the tumour is also a feature of the histology of the developing tooth.

The tumour possesses the power of eroding hard bone, a property which it shares with infiltrating neoplasms, e.g., rodent ulcer, derived from the basal cells of the epithelium.

Although it is recognized that adamantinomata occasionally arise in the region of the sphenoid, i.e., in bone only indirectly connected with the jaw, it is difficult to put forward any view as to the manner in which an epithelial neoplasm differentiated so definitely along the lines of the embryonic enamel organ originated deep to the periosteum of the lower end of the tibia.

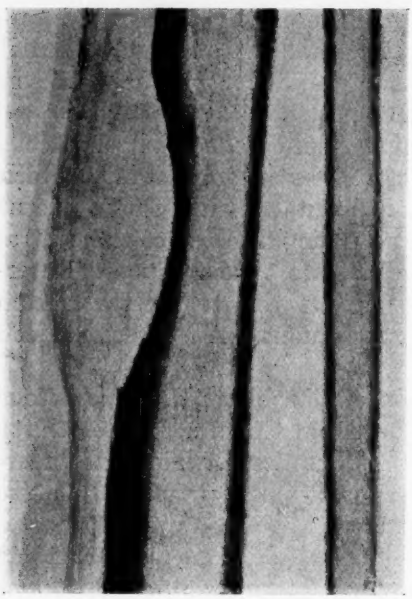

FIG. 323.—Radiogram of Fischer's case of primary adamantinoma of the tibia, lateral view. Compare excavated appearance of the eroded bone with that shown in *Fig.* 319. (*By courtesy of the 'Frankf. Zeits. f. Pathol.'*)

The only case of primary adamantinoma of the tibia which we have been able to trace in the literature—and it presents a close parallel—is that recorded by Bernhard Fischer[1] in 1913 :—

A healthy man, age 37, slipped on an iron staircase and struck his left leg against one of the steps. Severe pain and bruising resulted, but passed off in a few days. Five months afterwards at the site of the injury he noticed a swelling, which, treated by local applications, increased in size and became very painful. On examination four months later there was found between the middle and lower thirds of the tibia a smooth swelling, nearly the size of a hen's egg, and tender on palpation. Radiological examination (*Fig.* 323) showed the presence of a tumour at this site. Resection of the involved portion of bone was performed and an osteo-periosteal graft from the sound leg inserted. Eight months after the operation the patient was in good health and free from evidence of recurrence. Microscopically the tumour presented the characteristic picture of adamantinoma, with a spindle-celled stroma which in parts had undergone myxomatous degeneration.

Fischer, in discussing the mode of origin of the growth, discounts the possibility of a cell-rest composed of true enamel organ germ; and also of the transference of developing enamel organ from the oral cavity of the embryo to its tibia ("Man müsste denn annehmen, dass der Embryo zu der Zeit, als die Schmelzkeimanlagen entstanden, intrauterin in sein linkes Bein gebissen hat und dadurch einen Schmelzepithelkeim in die Tibia hinein befördert

hat. Aber diese Annahme dürfte zu absurd sein, um näher diskutiert zu werden.") In his opinion the condition could be accounted for by an excessive development of the multipotent embryonic ectoderm of the leg, with, in early embryonic life, its differentiation in the direction of enamel formation.

In the present case the epithelium must almost certainly have been subperiosteal from embryonic life: it would be difficult to account for its deposition subsequently by means other than a cutaneous wound, with implantation of a fragment of skin, or metastasis from a primary tumour elsewhere; of these there is no evidence. The possibility that the tumour might be teratomatous in nature would seem to be excluded by the entire absence of tissues other than those described above. The epithelium presumably remained of primitive basal-celled type, stimulated into tumour growth by the first injury, the second injury merely drawing attention to the presence of the condition by the onset of pain. For an explanation of such tissue dystopia we can only look to the embryologist.

REFERENCE.

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THE COLLOID ADENOCARCINOMATA OF THE BLADDER VAULT ARISING FROM THE EPITHELIUM OF THE URACHAL CANAL:

WITH A CRITICAL SURVEY OF THE TUMOURS OF THE URACHUS.

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INTRODUCTION.

It is little recognized by surgeons, pathologists, or even urologists that tumours may occur at the bladder apex which, while presenting themselves clinically and cystoscopically as simple epitheliomata or papillomata, display when examined microscopically many of the characteristics of adenocarcinomata of the rectum. These neoplasms have as a rule undergone colloid—or, better, mucoid—degeneration, but still show clearly ducts and acini containing mucin and lined by columnar epithelium interspersed with so-called goblet cells. This phenomenon has often puzzled both the pathologist and surgeon concerned. The condition is usually explained by a vague reference to embryonic rests of the 'allantois' or urachus, after the preliminary hypothesis of metastasis from the bowel has been found untenable. Sufficient material is now available on the clinical side to show that these tumours as described form a very definite entity, being all similar in their development and histological characteristics. The question of etiology may be largely solved by a more exact study of the anatomy and pathology of the urachus. This enables us to follow the origin and progress of the neoplasms in a regular and progressive way seldom attained in such a study.

The subject will well repay consideration. It gives a remarkable insight into the potency residing in primitive cells, and the conditions under which they take on malignant characteristics. It is, therefore, not without value in the study of cancer in general. On the other hand, it is by no means devoid of practical importance. Adenoma or adenocarcinoma is probably the commonest tumour of the bladder apex. In addition to the personal one to be described, seventeen definite cases have been collected from the literature, many of them with such good and full descriptions and illustrations that the structure, clinical march, diagnosis, and treatment of the neoplasms can be laid down with exactitude. These cases, scanty in number, represent in no way the frequency of the disease. Several personal communications of unpublished cases have been received from medical confrères. It is remarkable how the publication of a hitherto unexplained phenomenon will bring an increasing number of reported cases in its train. The importance lies in the consideration that what presents itself under the guise of a bladder tumour is in reality not a bladder tumour at all, and any attempt to treat

it as such by the usual methods—fulguration, radium, etc.—is predestined to failure. The early recognition of the true condition offers the patient at least a hope of effective cure by surgical means.

The plan to be followed in this paper will be in *Part I* to fix the reader's attention by the report of a typical case at a fairly advanced stage, to consider briefly the anatomy of the tract involved, and to follow this by a study of the neoplasms as a group; and in *Part II* to give the results of some studies of the pathology of the urachus, in order to explain the etiology of the growths and to classify them.

It should be remembered that no part of the bladder has an absolute monopoly of any particular pathological condition. The twofold origin of the viscus and its epithelium from mesodermal (Wolffian) and entodermal elements is only faintly reflected in its pathology. Papillomata of the ordinary type may appear, though rarely, at the apex; and adenomata or adenocarcinomata with or without mucin formation or degeneration may occur in any part of the organ without relation to the urachus. A true tumour of the urachus does not necessarily appear at the site of this structure's insertion into the bladder. The growth may have tracked along outside the mucosa and appear on the posterior wall and excentrically. This is rare, and such a case cannot be accepted as a urachal tumour unless its continuity with and origin from the urachus can be demonstrated. On the other hand, the studies of Störck and Zuckerkandl,[115, 116] Albarran,[3] and others have clearly shown the metaplasic potentialities of vesical epithelium, which can pass through all the stages of cystitis cystica to that of fully formed colloid adenocarcinoma (*see Fig.* 339). Indeed, in many cases the theory of urachal or allantoic rests has been unnecessarily dragged in to elucidate phenomena explicable along other lines. One case, not yet reported, of colloid cancer of the base of the bladder occurred in my practice. It was accompanied by so-called pseudomyxoma peritonei. The latter feature was no doubt unique, but the primary entity can easily be paralleled in the literature.

So definite, however, are the characteristics of apical vesical tumours of urachal origin, that there is no difficulty in differentiating them from the colloid cancers of the bladder wall itself, even when the latter occur towards the apex. The case of Kaltenbach[63] illustrates this point. The pedicle of a huge mass of tumour, resembling in microscopical structure a colloid cancer of the rectum, arose from near the apex. The structure of this growth was typically that of a bladder adenocarcinoma, arising from the mucosa and definitely pedunculated—a characteristic foreign to the urachal variety, which commences in the muscular wall and only subsequently penetrates the vesical cavity.

PART I.

CASE REPORT.

Mrs. B., female, age 54. Married, three children. Examined on Feb. 13, 1924.

HISTORY.—For ten years has had intermittent attacks of scalding and pain on micturition, with frequency. These symptoms have been more continuous for the last six months. Recently two attacks of hæmaturia without clots. Feeling of pain and 'bearing down' in the region above the symphysis pubis. General condition good. Has not lost weight or appetite.

ON EXAMINATION.—

General.—Well-nourished, healthy-looking woman. General examination elicited nothing of note.

Local.—Slight dullness above the symphysis pubis for one inch, in and close to the middle line. Tenderness and a sense of resistance on deep pressure downwards and backwards towards the bladder. No tumour palpable. No cord palpable between the navel and bladder.

Vaginal Examination.—The bladder was tender. Nothing definite to be felt.

Urine.—Hazy, stale, and offensive, of the type rarely found except in cases of diverticulum or tumour of the bladder. Red blood-cells, pus cells, and staphylococci with *B. coli*.

CYSTOSCOPIC EXAMINATION.—Mucosa generally inflamed, but not so as to obscure the vessels. At the apex and slightly excentric towards the left side was a shallow ulcer, circular, and 1½ cm. in diameter (*Fig.* 324). The surface was irregular, reddish on the whole, but with some white necrotic material in the centre. The edges were slightly raised, but did not appear to be indurated. There was no undercutting. The ulcer bled freely at the touch of a ureteral catheter and was painful. A piece was removed from the edge for pathological study.

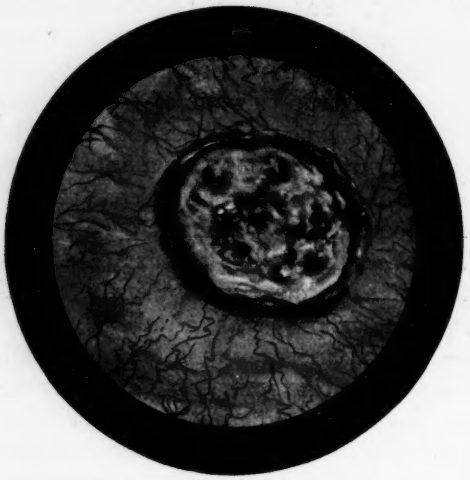

FIG. 324.—A cystoscopic view of the tumour.

Pathology of Tissue Removed.—"Tissue is highly vascular. The stroma is diffusely infiltrated with indefinite acinar forms. The cells lining the acini are of the high cylindrical type with nuclei at the base. Many are of the goblet variety. The tissue resembles an adenocarcinoma of the rectum" (Dr. Lynch).

The usual methods were used to exclude the possibility of a primary growth elsewhere.

OPERATION.—Before handling the bladder the peritoneum was opened from the umbilicus downwards and the whole abdominal cavity and bowel searched by touch and vision. Nothing abnormal was found. The urachus was isolated and appeared to run into a small, hard nodular growth at the bladder apex. The peritoneum was tacked down to this growth behind and was whitish and fixed, but the tumour had not ruptured through it. The whole mass, including the urachus and all the tissues between the transversalis fascia and the peritoneum, was removed together with the upper half of the bladder.

AFTER-HISTORY.—Quick convalescence. All urinary symptoms except frequency due to the reduced size of the bladder disappeared.

Eighteen months after the operation the patient was examined. The bladder had reached normal capacity. The urine was amber-clear. There were no pathological elements. Faint scar detected with the cystoscope at the site of resection. Nothing to be felt in the suprapubic region or pelvis. No urinary disturbance or complaint of any kind.

Two and a half years after the operation the bladder was still normal. There was no urinary trouble. A mass could be felt in the pelvis by bimanual examination just inside the left vaginal fornix.

Four months later during my absence she consulted a surgeon who, unfamiliar with the previous history, opened the abdomen and found the whole space between the transversalis fascia and peritoneum filled with recurrent growth. The glands of the pelvis on the left side were also involved. He contented himself with removing a piece of the tumour for examination. The patient died a few months later, almost

three years after the operation, without having had any recurrence in the bladder itself.

Specimen Removed (*Fig.* 325).—The tumour is cone-shaped, covered on one aspect with peritoneum, and attached to and perforating the bladder wall, forming there a circular ulcer between 1 and 2 cm. in diameter. A ring of the complete wall of the bladder, including mucosa, is attached to the specimen, giving a margin of several centimetres of healthy mucous membrane around the ulcer. On section there is seen a hard coagulated greyish jelly-like substance veined with pale red lines. Towards the lower part the appearance is more compact.

Microscopical Examination (*Figs.* 326, 327).—There is an indefinite musculofibrous condensation limiting the tumour above; below, the latter is in direct contact with the bladder muscle. There is a thin-walled stroma of vascular fibrous tissue throughout. Between the strands of this stroma are masses of irregular cell columns and acini. The latter are lined with a single layer of high cylindrical epithelium with basal nuclei. The acini are full of a homogeneous material that takes strongly a basic stain.

Fig. 325.—Semidiagrammatic view of the specimen removed, showing the ulceration of the mucosa of the bladder and the relation of the urachus to the tumour. The peritoneum and prevesical fat can be seen.

Fig. 326.—Low-power view of a section taken close to the bladder mucosa. The acini are in direct contact with the smooth muscle fibres.

Fig. 327.—High-power view. Goblet cells are numerous, but the epithelium is not so regular as in rectal growths. Some of the columnar cells are based on a row of cubical ones.

There are many goblet cells and others with mucin granules. Towards the bladder mucosa the acini are perforating and invading the bladder musculature, and may be seen among the individual fibres. At the upper part of the tumour the acini formation is less distinct. Many of the cells have undergone complete degeneration, and lie in unlined spaces filled with mucin. This substance appears also in the meshes of the stroma itself. The tumour is a columnar-celled carcinoma resembling the type frequently seen in the rectum.

The piece of growth removed from the metastatic mass a few months before the patient's death showed a similar structure, but with less colloid degeneration.*

To avoid redundancy, a more detailed description of the microscopic structure is reserved until a general survey has been made of all the cases reported. Prior to that it is necessary to summarize briefly the anatomical features of the urachus. A complete study on this subject has already been published.[12]

THE ANATOMY OF THE URACHUS.

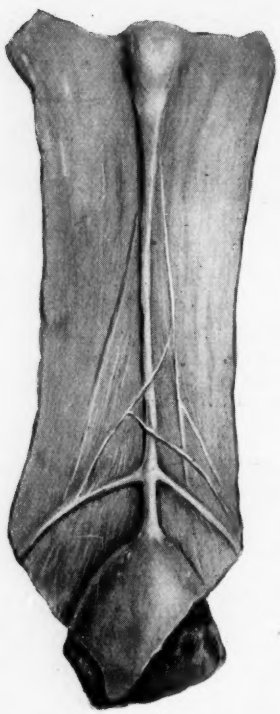

FIG. 328.—Drawing of the urachus and ligamentum commune of a child of three years as seen from the peritoneal aspect. The apex of the urachus is indicated by the point of junction of the umbilical arteries. (*See also Figs.* 349, 350.)

The tumours of the urachus at the bladder end, though frequently not recognized as urachal tumours at all, are by far the most important clinical group. To get a comprehensive view, however, it is necessary to consider the urachus and its adnexa as a whole, as faulty misconceptions on this subject have led many writers to draw unwarranted conclusions. The urachus lies in the space of Retzius. Considered in its widest sense, the latter embraces all that potential space bounded anteriorly from below upwards by the posterior and upper aspect of the symphysis pubis and the transversalis fascia; behind, by the bladder as far as its peritoneal reflexion, and above that by the peritoneum.

Ideally, though never actually, it forms an isosceles triangle, whose lateral boundaries are the obliterated umbilical arteries and whose base is formed by the puboprostatic ligaments below. The apex of the triangle is the point where the two umbilical arteries and the urachus meet above. This point is, at birth, the umbilicus, but as the urachus descends immediately, it is much lower down in the adult, being situated as a rule at the junction of the lower with the middle third of the distance between the bladder apex and the umbilicus (*Fig.* 328, 329). As there is more or less union between the umbilical arteries and the two structures between which they lie—namely, the peritoneum

* The term 'colloid' is in such constant use that it is not possible to avoid it in connection with these growths. It must be remembered, however, that the material formed is, strictly speaking, mucin and not colloid.

behind, and the transversalis fascia in front—the space is closed laterally. Lower down it passes deeply over the sides of the bladder into the pelvis. With this portion, however, we are not concerned.

The urachus descends, and as it is attached on each side to the umbilical arteries, the latter are drawn together and form one continuous band passing from its apex to the umbilicus. The name of *ligamentum commune* has been suggested by Professor Peremeschko[98] for this structure (*Figs.* 328, 329, 331). It consists merely of the drawn-out adventitial muscular and possibly elastic tissue derived from the walls of the obliterated umbilical arteries. It may give rise to the mesoblastic tumours which when they occur in this region have been erroneously attributed to the urachus.

On the assumption that it normally extends as far as the navel, it has, indeed, been the fashion to attribute to the urachus almost any pathological condition occurring in the upper part of the space of Retzius. Tumours, however, may have their origin from any of the structures, fibrous, fatty, or vascular, above, or from the bladder itself below, and in both cases may involve eventually every part of the space, working down to and invading the bladder from above, or passing up from it towards the umbilicus. With many of these, the urachus has no connection.

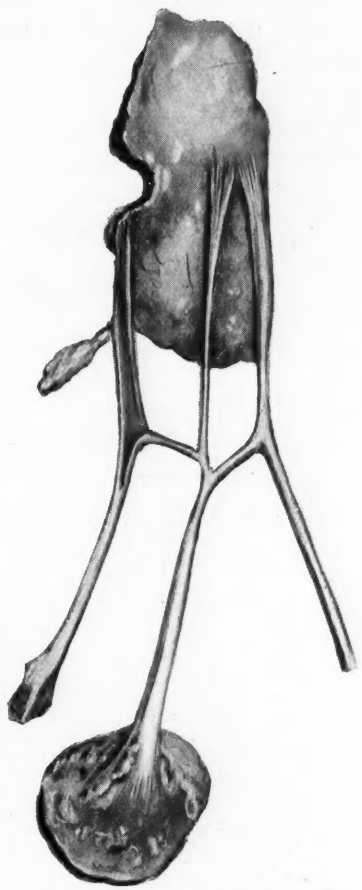

FIG. 329.—A dissected specimen of the urachus and attached ligaments from an adult. Note the splitting of the left umbilical artery.

Divergences from the Ideal Anatomical Relationships.—It must be taken into consideration that the actual anatomical conditions which prevail are not the ideal ones described above.

There is as a rule, it is true, the central ligamentum commune, but frequently the umbilical arteries are so teased out by the descending urachus that innumerable fibres of their adventitia are spread in a fan-shaped manner, sometimes over an area as wide as three inches (*Figs.* 328, 330). One or both of the umbilical arteries may be avulsed completely and lie free, being only connected to the umbilicus by fine strands of adventitial tissue. Thus, instead of one space, many potential ones exist, bounded by the strands of this so-called Luschka's plexus. Again, the urachus is frequently not central. It may have lost its attachment to one umbilical artery, and its upper end may be pulled laterally and at the same time in a downward direction by the other, so that it takes a course from the bladder apex upwards and outwards to the right or left (*Fig.* 330). *Urachal tumours are accordingly not always in the middle line.* Even where it is laterally displaced, however, the urachus is firmly attached by fibrous strands to the umbilicus so that it

can carry out its musculo-tendinous function of steadying the bladder during micturition.

Rests from Urachus left during its Descent.—In previous papers[12, 13] I have suggested that the urachus during its descent may be pulled asunder or that a portion may be detached, thus leaving some of its cellular content behind, and that from these rests epithelial tumours may arise. These tumours may be found at the umbilicus itself, or between that structure and the apex of the urachus 10 or 11 cm. below. Certain adenomatous structures and neoplasms occurring in this region are referred to later (*see Figs.* 349, 350).

FIG. 330.—A drawing of an adult urachus to illustrate how the structure may be pulled over by losing its attachment to one of the umbilical arteries.

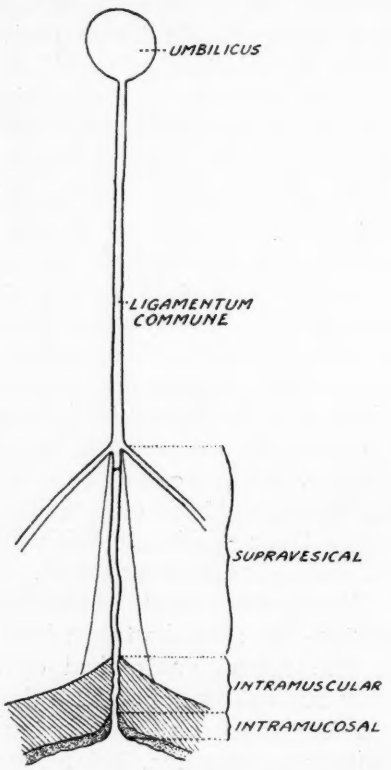

FIG. 331.—A diagram to show the subdivisions of the urachus.

Divisions of the Urachus.—The urachus is a structure about 5 to 6 cm. in length, and for our present purpose can be conveniently divided into three portions (*Fig.* 331): (1) Supravesical, where it is entirely above the bladder; (2) Intramural, where it lies in the muscular wall; (3) Intramucosal, in cases where its lumen is in direct communication with the bladder cavity. In the 66 per cent of cases where there is no communication, there are only two portions to be considered, namely, supravesical and intramural. In all three parts the canal is patent and lined by many layers of epithelial cells (*Figs.* 332–335).

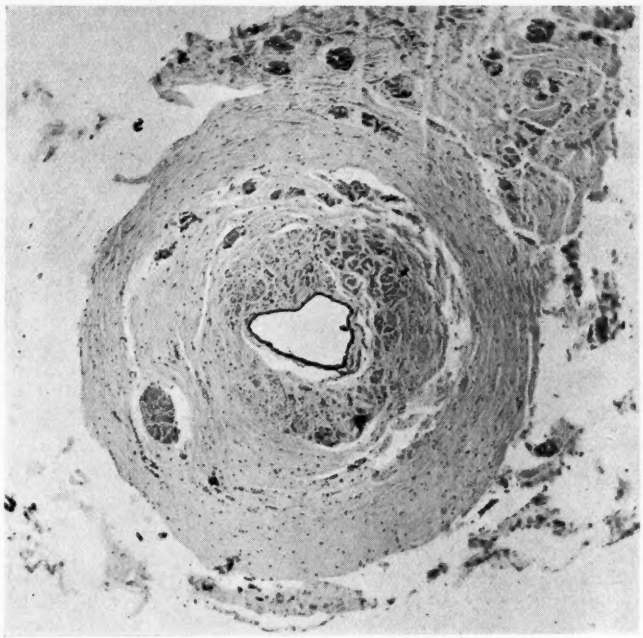

FIG. 332. — Low-power view of a urachus. The specimen was taken from a male adult, 1·5 cm. above the bladder.

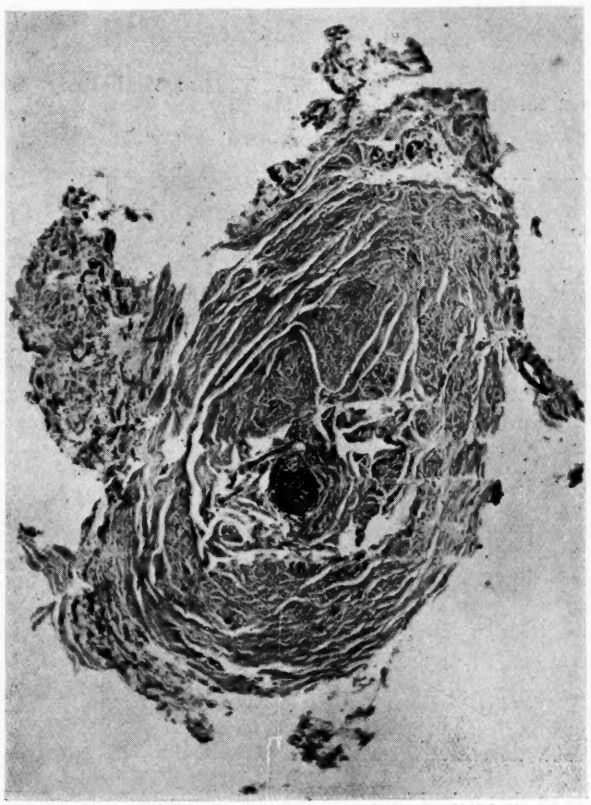

FIG. 333.—Section through the apex of the urachus, 5 cm. above the bladder. The epithelium forms a solid column.

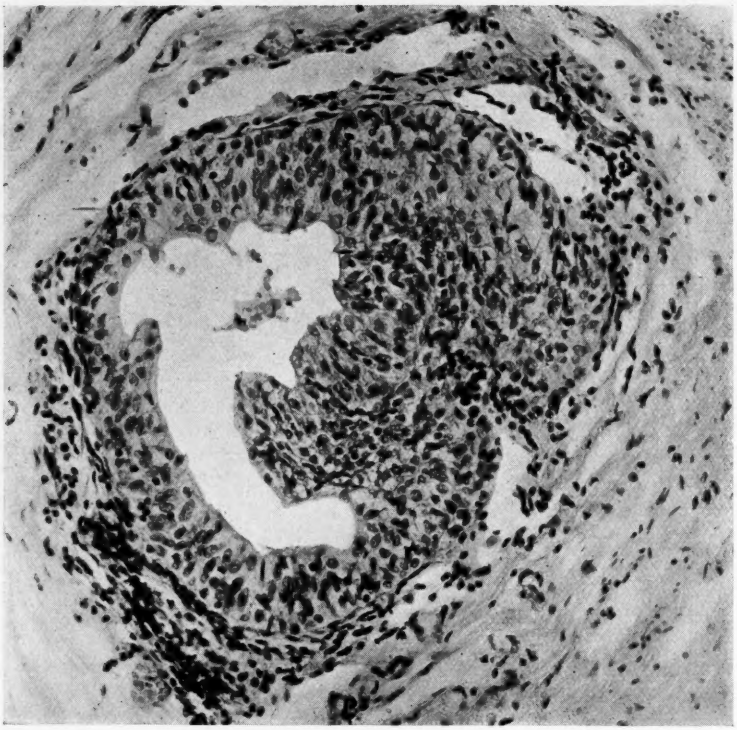

FIG. 334.—Section of an adult urachus 2 cm. above the bladder. The irregular heaping up of the cells is well shown.

FIG. 335.—The same urachus as shown in *Fig.* 334, 4 cm. above the bladder.

That centimetre of the urachus which penetrates the muscular wall of the bladder is the most important from the pathological point of view, for it is usually at the upper part of this, or in the lower end of the extravesical section, that new growths originate (*see Figs.* 337, 338, 340).

In some cases where the canal communicates with the bladder there is a wide trumpet-shaped opening forming a diverticulum (*Fig.* 336 A). This probably existed in the cases of Michin,[83] Randall,[99] and Lavenant,[74] where the growths were seen to project from a funnel-shaped retraction of the bladder apex. In some instances the opening is at the top of a papilla (*Fig.* 336 B), and at others it is flush with the bladder mucosa. Neoplasms in the former would tend to project into the bladder, and in the latter appear as a flat ulcer. Both these types have been found.

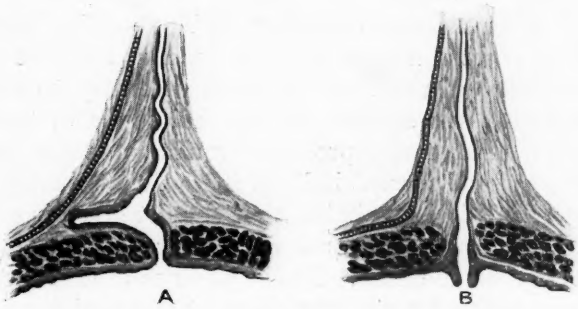

FIG. 336.—Two methods by which the urachus may connect with the bladder. In A there is a wide expansion; in B the canal opens on the summit of a papilla.

Unformed and Undescended Urachus.—In a previous paper on umbilical fistulæ of urachal origin,[13] it was shown: (1) That in some cases the urachus does not form at all—that is to say, the upper part of the bladder fails to narrow at the appropriate stage of fœtal life, and as a result its apex remains at the umbilicus, no descent having taken place; (2) That in other cases the urachus is not properly differentiated from the bladder, the cavity of the latter being continued by a wide canal as far as the umbilicus. All intermediary stages of formation and descent may be encountered, the apex of the bladder reaching in some instances well above the symphysis. In these the urachus is usually normal, but placed at a higher level. This fact is of some importance, for a tumour which is really in the intramural part of the urachus may be palpated well above the symphysis, as in the cases of Michin[83] and Schwarz.[110] Finally, in the extreme case of ectopia vesicæ, the urachus and the bladder remain open anteriorly and the apex of the combined organs is at the umbilicus.

CLASSIFICATION OF TUMOURS OF THE URACHUS.

Classification according to Position (*see Fig.* 331).—Tumours of the urachus may be divided into those:—

1. Of the intramucosal portion, where this exists (33 per cent of cases).
2. Of the intramural portion.
3. Of the supravesical portion, which extends upwards 5 or 6 cm. from the apex of the bladder.
4. Of abnormal urachal rests above the apex of the urachus and between this and the umbilicus.
5. Of an unformed or undescended urachus. In the former case the neoplasms, strictly speaking, belong to the upper part of the bladder.

6. Of the fibrous and muscular strands derived from the umbilical arteries above the true apex of the urachus. They are not tumours of the urachus at all, but it is convenient to specify them here, as many of the cases reported as urachal neoplasms belong to this group. Microscopically they contain no epithelial elements. They are fibromata, fibromyomata, or sarcomata.

7. Of the upper part of an ectopic bladder, which is in reality the anlage of the urachus.

Classification according to Type.—This classification is based on a study of all cases of neoplasm of the urachus found in the literature. A large amount of sifting of material has been necessary to exclude those reports where the evidence was against the urachal origin of the neoplasm described. On the other hand, many probable urachal tumours lie buried under other captions. The help of current indexes is no doubt inadequate to disinter some of them, so that it is improbable that the list given is complete ; it provides, however, sufficient material for a satisfactory analysis. One variety of neoplasm calls for special mention. In the absence of undescended urachus, tumours derived from urachal rests may form at the umbilicus itself. Apart from this theoretical possibility, some of the actual growths in this region resemble so exactly proved urachal tumours that there is little doubt as to their true nature. The whole subject of adenomatous growths of the umbilicus is, however, so extensive that its inclusion would complicate unnecessarily the problem to be considered. Apart from casual mention and a limited reference to the literature in the bibliography, it will not be dealt with (*see* p. 461).

Rejection of certain Cases from the Category of Urachal Growths.—Six cases should, I think, be excluded from any future statistical study of the subject. As they are repeatedly used to illustrate urachal pathology, short notes and comments are given of each of them. The case of Pinheiro[96] said by Kielleuthner to be one of carcinoma of the urachus was not available to me in the original, and is accordingly not included in this study. The other six are as follows :—

1. Klopp's case.[67]

Klopp described in 1921 as sarcoma of the urachus a case (male, age 50) in which a small collection of mononuclear cells accompanied by a giant cell were found in connection with a canal lined by squamous epithelium discharging at the umbilicus. The evidence that this was a tumour of the urachus, or indeed a tumour, at all is unsatisfactory, though the fact that the sinus reached down as far as 12½ cm. from the umbilicus might relate it with the apex of the true urachus. Mononuclear and giant cells have been found in simple cases of umbilical fistula.

2. Rankin and Parker[100] (*Case* 7 in reference cited).—

This was apparently a mixed tumour of small size in a male, age 68. It was a squamous-celled carcinoma with a few irregular spaces lined by epithelial cells. It was said to be attached to the urachus, which was discharging at the umbilicus. It is, however, very easy to confuse the thick strand of the ligamentum commune with the true urachus. As is well known, most of those interesting cases with a pus discharge at the umbilicus do not concern the urachus at all, and merely track down for a few centimetres in the abdominal wall. In the same article the authors mention three other undoubted cases of urachal tumour.

3. Göbel's case[43] (published in 1905).—

Male, age 40. A cancer, originating in a lesion caused by *Schistosoma hæmatobium*, had perforated the anterior wall of the bladder and finally ruptured through

the abdominal wall at the umbilicus and at another place farther down. All tumours extending in the space of Retzius, however, tend first of all to rupture through the skin at the weak point represented by the umbilical scar. The apex of the urachus is normally 11 cm. below this, and there seems to be no reason to think that this structure was involved. The case was published as one of a very long series in which carcinoma had supervened on bilharziasis. In the main, the growth had the characteristics of a squamous carcinoma.

4. HOFFMANN'S case[57] (published in 1870).—

Male, age 28. This was said to be a carcinoma in a patent urachus involving the bladder. A careful study of the case shows that the bladder had never left the umbilicus and the urachus had not formed. The narrowed apex of the bladder reached the umbilicus, and starting from this point the organ became involved in a squamous epithelioma. There is no evidence that this was primary in the bladder, and it may easily have originated from the deep surface of the umbilicus, from ectodermal elements included in the umbilical scar at the time of its formation, or from some other rest in the neighbourhood. Possibly it arose from the vesical mucosa, as squamous epitheliomata are not very uncommon in the bladder.

5. FISCHER'S case[36] (published in 1893).—

Male, age 32. The patient is said to have had a patent urachus till the age of three, with urine discharging at the umbilicus. A carcinoma developed in the region of the navel and ruptured through the skin. The nature of the carcinoma is not stated. At the autopsy it was found that the growth had involved but not penetrated the wall of the bladder at the apex. The latter was in the normal position. The urachus had thus formed and descended, and the case was probably one of carcinoma arising in the neighbourhood of the umbilicus and involving the bladder by direct downward spread in the space between the peritoneum and the transversalis fascia.

6. LONG'S case.[78]

Long published a case in 1927 in which he found some hairs in a cyst at the umbilicus. He described it as a dermoid cyst of the urachus. The evidence produced in support of this diagnosis is not completely convincing.

Adenocarcinoma and Colloid Cancer Arising in the Upper Part of Ectopic Bladders.—As already pointed out, the upper part of the ectopic bladder is the anlage of the urachus, and reference to adenomatous structures in that region is of passing interest.

Six cases at least have been reported. In those of Zuckerkandl,[134] Ehrich,[32] and Hager,[48] the colloidal element predominated, while those of Scholl[108] (*Case* 5 in reference cited), Gerahty,[40] and Störck[115, 116, 34] were simple adenocarcinomata of the intestinal type.

ANALYSIS OF CASES OF URACHAL TUMOURS IN REGARD TO TYPE AND POSITION.

Including the author's, 44 cases are given in the table on p. 434. Ten of these are mesoblastic tumours which, though not considered to be urachal neoplasms, have been published as such. The 44 are divided as follows:—

Fibro-adenoma	3
Adenocarcinoma (not colloid)	4
Colloid adenocarcinoma	19
'Mixed tumours' (including those of unusual pathology)	8
Fibroma and fibromyoma	4
Sarcoma	6
Total	44

The determination of the nature of the cases does not rest entirely on the individual author's opinion, but on a very careful study of the individual descriptions and a comparison of these with each other, having in mind the true anatomy, morbid and systematic, of the urachus. The reports cover a fair period of time, and vary considerably in completeness. It appears probable that fibro-adenoma, adenocarcinoma, and colloid cancer result progressively from certain changes in urachal cells which can be studied in a large series of specimens taken from the post-mortem and dissecting-room, and they are accordingly grouped as one series.

The term 'mixed tumours' denotes a group presenting unusual features. They contained in some instances both sarcomatous and carcinomatous elements, and had a tendency to cystic or acinar formation. They were all connected with the apex of the bladder, and were in some instances called teratomata and attributed by the authors reporting them to 'allantoic' rests.

The ten cases of fibroma, fibromyoma, and sarcoma arose from non-epithelial structures, and when the position of the tumours is analysed it will be seen how strikingly this class is confined to the region of fibrous bands and teased-out arterial adventitia above the level of the urachus. There is of course no reason why such tumours should not arise from the same elements in the urachus itself, but as a matter of experience urachal tumours seem invariably to contain epithelial elements.

The table annexed gives an excellent impression of the situation of the different examples.

CASE INCIDENCE OF DIFFERENT VARIETIES OF URACHAL TUMOURS.

CLASS OF TUMOUR*	COLLOID CANCER	ADENO-CARCINOMA	'MIXED TUMOURS'	FIBRO-ADENOMA	SARCOMA	FIBROMA AND FIBRO-MYOMA	TOTAL
I, II	5 (*a*)	2 (*d*)	1 (*g*)	1 (*k*)	—	—	9
III	13 (*b*)	1 (*e*)	6 (*h*)	2 (*l*)	—	—	22
IV	1 (*c*)	1 (*f*)	1 (*i*)	—	—	—	3
V	—	—	—	—	—	—	0
VI	—	—	—	—	6 (*m*)	4 (*n*)	10
Total	19	4	8	3	6	4	44

*I = Intramucosal. II = Intramural. III = Supravesical. IV = Tumours derived from urachal rests. V = Tumours arising in an unformed or undescended urachus. VI = Tumours not involving the bladder and arising from adventitial and muscular structures in the space of Retzius.

Key to reporters of cases indicated in table :—

a. Author's case, Kielleuthner,[66] Zuckerkandl,[134] Scholl[108] (*Case* 4), de Korte.[29] *b.* Chute and Crosbie,[26] Störck and Zuckerkandl[116] (two cases), Lavenant,[74] Barringer,[10] Schwarz,[110] Randall,[99] Green,[45] Scholl[108] (*Case* 3), Rankin and Parker[100] (*Case* 5), Khaum,[65] Michin,[83] Pendl.[93] *c.* Hue and Jacquin.[59] *d.* Hartmann,[50] Scholl[108] (*Case* 1). *e.* Scholl[108] (*Case* 2). *f.* Koslowski.[69] *g.* Blavet di Briga.[15] *h.* Rotter,[103] Rankin and Parker[100] (*Case* 6), Lenormant,[77] Nuboer,[89] Cullen,[28] Doiran.[30] *i.* Adám.[1] *k.* Hagner.[49] *l.* Nicholson,[87] Hector.[52] *m.* Greig,[46] Frank,[38] Villa,[122] Greig Smith (two cases),[113] Vöncken and Cambresier[125] (*Case* 2). *n.* Brady,[18] Rankin and Parker[100] (*Case* 4), Aveling,[9] Vöncken and Cambresier[125] (*Case* 1).

Remarks on the above Table.—*Class VII*, relating to growths in cases of ectopia vesicæ, is not included. Under *Class V*—tumours in an unformed urachus—not a single example occurs. Hoffmann[57] and Fischer[36] were inclined to place their cases under this heading. They have been referred to above and rejected from the category of urachal neoplasms.

No distinction is drawn between intramucosal and intramural urachal growths. The former would be expected to be rare, as from the anatomy of the urachus they could only occur in 33 per cent of the total—the percentage of cases in which the urachus communicates directly with the bladder cavity. Such growths would appear as bladder tumours and be mistaken for them, just as a growth projecting into the duodenum from the ampulla of Vater might commence so near to the mucosal junction as to be indistinguishable on the basis of situation alone from a tumour of the duodenal mucosa itself. Kielleuthner[66] thought that his case came under this category, but a study of his description and illustrations shows the origin of the tumour to have been intramural.

The distinction between the intramural and supravesical tumours is a real one, the pathology and symptomatology being different to some extent. The line is, however, hard to draw between borderline cases. The early malignancy and rapid onset of severe bladder symptoms characterize the intramural variety. The determination was made by considering the march of the symptoms and the nature of the growth as found at operation or autopsy; also the evidence of the age of different sections estimated from the advance of degeneration. The name of the author is given as sufficient indication to enable the reader to locate in the bibliography the report where the full description of the case will be found. These cases vary from the least advanced one of Kielleuthner,[66] in which the tumour was confined to the bladder wall, to that of Pendl,[93] in which a huge growth occupied a large part of the space of Retzius and reached almost to the umbilicus.

The preponderance of the colloid type over all other varieties, when one considers merely those tumours which arose from the part of the urachus within the bladder wall or immediately above, will appear from the following figures of recorded cases:—

Colloid adenocarcinoma	18
'Mixed tumours' of unusual pathology..	5
Adenocarcinomata without colloid degeneration	3
Fibro-adenomata (2 discovered incidentally on post-mortem examination)	3
	29

Not a single one of the cases penetrating the bladder mucosa was of the mesodermal type.

Pathological Study of Colloid Carcinoma of the Urachus involving the Bladder Apex.—With the exception of those of Störck and Zuckerkandl, all the eighteen cases referred to under (*a*) and (*b*) in the table on p. 434 were reported between the years 1911 and 1930. The tumours are all essentially similar in microscopical appearance, and doubtless arose from the tiny adenomatous structures which are such a striking feature of the lower end of

the urachus as studied in routine post-mortem specimens (*Figs.* 337, 338). Many of the fine details described will become significant when compared with the latter. The growths found clinically represent the expected end-results of processes observable in the apparently normal urachus.

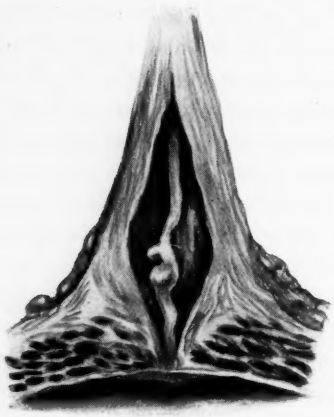

FIG. 337.—The urachal canal dissected from its sheath, showing two small cysts at its lower end.

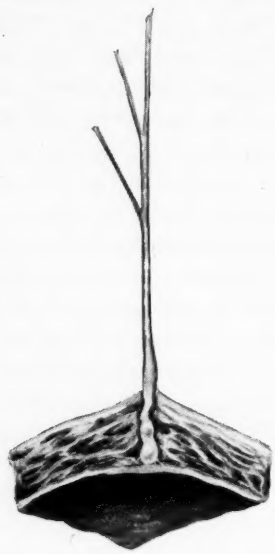

FIG. 338.—Urachus ending blindly at the mucosa, and dilated into a small cyst at this point. From a female 56 years of age.

Distinction between the Urachal Tumours and other Adenomatous Growths in the Bladder.—Lendorf[76] demonstrated that in the embryo there were no downgrowths of the mucous membrane to form glandular structures, while in children these formations appeared as solid buds, and later in life as definite simple glands. In adults the latter may take on a more complex structure, and in cases of inflammation definitely secrete mucin, as exemplified in cystitis cystica. (*Fig.* 339.)

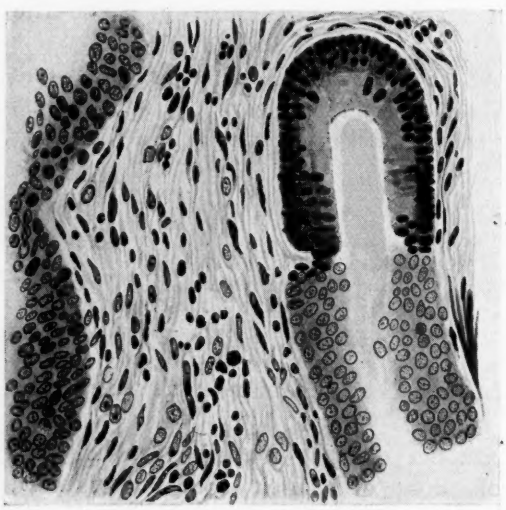

FIG. 339.—An illustration from Bayer's article to show the change of transitional epithelium into columnar. The section is taken from the bladder. It will be noted that the transitional epithelium has reverted to a primitive type (*see* p. 450).

(Compare also the work on this subject of von Brunn[124] and Albarran.[3])

The small multiple tumours present in cases of cystitis cystica become important pathological structures, and occur either singly or as multiple neoplasms arising from the bladder mucosa. These may take the form of simple adenomata, adenocarcinomata, or colloid growths. The structure of the latter may be similar to that of

colloid cancer of the rectum or to that of the urachal tumours which resemble it.

These pure bladder neoplasms may be differentiated from urachal ones in that they arise from the mucosa, tend to be pedunculated, and only penetrate the muscular wall in the advanced stage. The urachal tumours, on the contrary, originate at the apex of the bladder either in the muscular wall or completely outside it, and penetrate the mucosa later, or in a few cases merely bulge it.

Pure vesical adenomata of the simple type are exemplified in the cases of Albarran,[3] Astraldi,[7] Bridoux,[19] Cahen,[23] Carraro,[24] Cassanello,[25] Egger,[31] Herbst and Thompson,[54] Judd,[62] Lavaux,[73] Motz,[86] Notthafft,[88] Pavone,[92] Sacchi,[105] Watson,[129] and Uteau.[120] Instances of adenocarcinoma have been reported by Alexander,[4] Audry,[8] Bristow,[21] and Kanamori[54]; colloid cancers by Bayer,[11] Haake,[47] Kaltenbach,[63] Mandelbaum,[81] Pinesohn,[95] Posner,[97] Rauenbusch,[101] Rochet and Martel,[102] Sauter,[106] Schmidtmann,[107] Sharp,[111] Sperling,[114] Störck,[115] Zausch[133] (two cases), Zuckerkandl,[134] and Fontana.[37] In addition to the reports of the above authors the subject is discussed in the writings of Aschoff,[5] Blum,[16, 17] Frisch and Zuckerkandl,[39] Gerahty,[40] Paschkis,[90] Verhoogen,[121] von Limbeck,[126] von Brunn,[124] and Wägeler.[127]

'Mixed Tumours' of the Bladder Proper.—The type of tumour we have called 'mixed' in the study of urachal cases has its counterpart also in neoplasms arising from the bladder epithelium. These growths have, as a rule, been described as sarcocarcinoma, myosarcocarcinoma, etc. They are characterized by the presence of spaces or acini lined by secreting columnar or cylindrical epithelium, and have frequently a cystic nature when considered macroscopically. Lenormant[77] in his article discusses them briefly. Other examples that may be referred to are described by Albarran,[3] Clado,[27] Heineke,[53] Herczel,[55] and Krompevher.[70]

Adenomata Occurring in the Trigone and its Neighbourhood.—The discussion of this type of tumour opens up the question of aberrant prostatic glands. Certainly the pathology is different from that of adenomatous growths in the fundus, though the two varieties probably overlap. Case reports and discussions on this subject will be found in the articles of Goodall,[44] Hermann,[56] Horn and Orator,[58] Lubarsch,[79] Paschkis,[90] Thomas,[119] and Wittsach.[131]

SYSTEMATIC DESCRIPTION OF THE COLLOID ADENOCARCINOMA OF THE LOWER END OF THE URACHUS.

While all the eighteen cases cited are essentially similar in their microscopical structure, they may roughly be divided into two classes having regard to their clinical course, macroscopical appearance, and the exact method by which they originate.

Class 1.—This comprises the great majority, in which the upper part of the growth is encapsulated and extends well up into the space of Retzius, whereas the lower end which is in the bladder wall has no definite capsule, the acini lying in direct contact with the muscular tissue. Most of them have ultimately penetrated the mucosa, but the length of symptomless history as regards the bladder, combined with their large size and the presence of

calcification in the capsule and stroma, leads to the conclusion that they existed for a considerable time as simple tumours of the type described in the cases of Nicholson,[87] Hector,[52] and Hagner,[49] and took on frankly malignant characteristics late in their course.

Class 2.—This group is represented in the cases of de Korte, Albarran, Kielleuthner, that of the author, and possibly Scholl's *Case* 4. The neoplasms in this category apparently arise from microscopic adenomatous structures of the urachus, and become malignant at a very early stage. They are almost entirely within the wall of the bladder, and form cone-shaped tumours (*see Fig.* 325) with the apex above. There is no true capsule—at most a condensation of the tissues around the periphery. In the cases mentioned the growths were first discovered by cystoscopic or post-mortem examination, and no tumour was palpable. As in Class 1, however, the most degenerated areas were always those most remote from the bladder mucosa, suggesting that the point of origin was the upper part of the intramural portion of the urachus. Their pathological appearance is similar to that of the lowest portions of the first group.

In a few instances of the cases described under Class 1, the mucosa had not been penetrated at the time of examination. These no doubt all occurred in subjects in whom the lumen of the urachus reached only as far as the external surface of the bladder mucosa (*see Figs.* 337, 338). Such are the cases of Barringer and Pendl, and Störck and Zuckerkandl's *Case* 1.

As found at operation and subsequent examination or autopsy, the growths ranged from the size of a hazel-nut in Kielleuthner's case to that of an adult head in Pendl's. Two portions could be distinguished, an upper involving the whole or part of the supravesical urachus, and a lower incorporated in the bladder wall at the apex.

The capsule was in direct continuity with the muscular and adventitial wall of the bladder below, and ended there. Within the musculature of the bladder wall the cells invaded directly the surrounding tissues, lying in apposition with the muscular fibres, in some cases penetrating their sheaths.

The general consistency of the tumours was cystic, and they were composed in every instance of cavities lying in a network of stroma. These cavities varied in size from large ones with several ounces of content to tiny spaces only distinguishable under the microscope.

The upper part of the tumours lay between the transversalis fascia and the peritoneum in the anterior abdominal wall, near to the middle line. They were all adherent to the peritoneum towards the bladder end, and could not be removed without opening the peritoneal cavity. As a rule they were easily shelled out after severing the ligamentum commune, which attached them to the umbilicus, down to the point where they were adherent to the peritoneum. In more than one instance the growth had broken through the peritoneum, and in one had formed adhesions with the abdominal contents. In Pendl's case the tumour was adherent also to the transversalis fascia and the recti muscles.

From the upper part of the growth a firm cord-like structure may pass up towards the umbilicus (*see Fig.* 325). It may on section show the epithelially-lined canal of the urachus which has been pushed up by the development

of the growth in a superior direction. If the canal is completely involved in the tumour, the only connection with the umbilicus may be by means of the ligamentum commune or the strands of Luschka's plexus (*see Figs.* 329, 330). In cases where the urachus has not descended, the growth may reach as far as the navel, but as a rule falls short of it even then.

The Capsule.—The capsule is always fairly definite, but may be markedly irregular, corresponding to the lobulated form of the tumour itself. It may be so thin that the gelatinous content shines through. It may be as much as 3 cm. in thickness, and consist of a layer of fibrous tissue covering one rich in smooth muscle fibres. In advanced cases the fibrous bundles are old, sclerosed, poorly supplied with blood, and may have undergone hyaline or calcareous degeneration. In some instances they harbour foci of inflammatory round cells.

The apparent capsule is not true capsule in its whole thickness, the presence of alveoli in the inner layer demonstrating that this portion is merely a compressed part of the tumour proper.

As mentioned already, the outer sheath of fibrous and muscular tissue becomes continuous with the adventitial and muscle layers of the bladder, so that the capsule is always incomplete below.

The Stroma.—The stroma consists of bands of varying thickness, and may be concentrated into a fibrous mass towards the centre of the tumour, as in the case of Barringer. It may show all the changes of hyalinization and calcareous degeneration, and in parts may actually be infiltrated by mucin derived from acinous groups at one time existing in the meshes. It encloses spaces of varying magnitude from small microscopical ones to huge cysts. In many cases the mucin in these spaces is in direct contact with the fibrous tissue, though there may be traces of the original cell lining. At the bladder end the stroma takes the form of young or medium fibrous-tissue bundles lying irregularly among the acini of the tumour and the muscle fibres of the bladder. The latter tend to be replaced by the fibrous tissue (*see Fig.* 326).

The Epithelial Element.—So great is the output of mucin that rapid degeneration both of the cellular elements and the stroma takes place, and the undegenerated portion or growing edge is small. In all the cases reported, however, that section of the tumour immediately deep to the bladder mucosa invariably presented the structure in its early form, except in that of Pendl, where no active areas were discovered, and in that of Barringer, where the tumour did not penetrate the mucosa.

The macroscopical and microscopical aspect as seen in the region of the bladder will accordingly be considered next. The former depends to some extent on the anatomical structure of the urachus at this level in the particular individual concerned.

Cystoscopic Appearances.—

1. *Ulceration* (5 cases).—There was a very definite circumscribed ulceration, or rather appearance of ulceration, involving an area of 1 to 2 cm. in diameter (*see Fig.* 324). This area presented points of necrosis, and had a margin raised above the surrounding mucosa, which appeared to remain normal right up to the circumference of the ulcer. The latter was in some cases slightly excentric towards the right or left.

2. *Papillomatous Appearance* (4 cases).—The tumour appeared as a papillomatous area well defined and surrounded by normal mucosa. In one instance there was a grey granular circumscribed patch on the summit of an elevation which the pressure of the tumour had caused. In the cases of Lavenant and Michin there was a cone-shaped retraction at the apex of the bladder from which fronds of tumour emerged. These were evidently patients in whom the urachus had originally a trumpet-shaped dilatation at the point of communication with the bladder (*see Fig.* 336 A). In Green's case there was a small opening 3 mm. in diameter through which gelatinous material emerged.

3. *Bulging of the Bladder Wall.*—In 3 cases the mucosa was intact, or only corrugated through adhesion to the growth.

In the remainder the cystoscopic appearances are not clearly described.

Microscopic Characteristics.—Alike where the bladder defect is flat and where polypoid or papillomatous protrusions are present, the surface is covered with a layer of high cylindrical epithelium only interrupted at the points where necrosis has taken place or at the tips of the papillæ. In de Korte's case the surrounding transitional epithelium was becoming transformed into the columnar type. On this surface, according to Albarran, open the ducts of the glands which lie in the wall of the bladder. These take on complex and branched forms, and the acini lie directly among the muscle fibres. They are surrounded by young fibrous tissue which tends to replace the muscular elements and form cores for the papillæ which project into the bladder.

The cells lining the acini are of the high cylindrical variety with basal nuclei. They exhibit the various stages of the formation of mucin. In some it exists in the form of small granules, in others in droplets, and in others again these droplets have run together to form a central mass. In some places the defined free margin of the cell has ruptured and delivered its mucin into the acini. The secretion of mucin is great, and the quantity delivered in the urine very noticeable. Cells of the same type push into the fibrous framework, and are seen multiplying by an amitotic process. Large typically carcinomatous groups of cells may also occur in the stroma.

In many places there is more than a single row of cells, clearly apparent in my specimen, and described by most of the authors who have given attention to the microscopical detail. Instead of the cylindrical epithelium resting on the underlying stroma, it is based on another row or two rows of cells. The latter are cubical and flattened, but their axis is irregular. *The impression is given that the topmost row of a primitive transitional epithelium such as exists normally in the urachus has taken on the rôle of secreting columnar epithelium.* This is a most important indication of the real origin of the tumours. There is no membrana propria, the cells being in direct contact with the fibrous tissue of the stroma. The epithelium of the urachus has likewise no membrana propria, and its absence in the case of doubtful neoplasms is of considerable value in assigning the correct etiology. (*See Figs.* 334, 344).

Appearances in the Older Parts of the Growth.—The structures rapidly degenerate as one passes upwards from the bladder mucosa. Masses of mucin are seen lying free in spaces formed by fibrous tissue, and even apparently

among the individual fibres. In these masses may be seen 'ghosts' of degenerated cells, fragments of nuclei that still take the stain, degenerated red blood-corpuscles, and sometimes remains of fibrous cells. In some of the spaces, more especially the smaller ones, the process of degeneration is not so advanced. Rows of cylindrical cells adhering to each other line small sections of the wall of the cavity, or are tilted up from this at one end by the extravasation of blood between them and the lining membrane. Pale nuclei may still be seen here and there at the base. In other cases the epithelium has become flattened from pressure, and all grades are seen from low cylindrical, through cubical, to cells as flat as pavement cells. The cubical cells seem in some cases to represent the highest development of the urachal epithelium. *In the cases of Barringer and Pendl they were actively secreting.*

In some instances, and especially in the larger cavities, there is more than one layer of cells lining fragments of the wall. Ruptured alveoli, and alveoli lying back to back without intervening fibrous tissue, may also be seen. The whole field is saturated in mucin, not only in the cavities but between the fibrous bundles of the stroma. Large cells with vesicular nuclei may be seen penetrating the fibrous tissue. There are no formed blood-vessels except in the stroma, but a good deal of free blood in some of the larger cavities, giving a red jelly-like appearance to the content. Yellow streaks rich in cell membranes and débris can sometimes be distinguished from the surrounding white masses.

In Pendl's case the cells were all of the cubical variety. He likened the appearances seen to those in the older parts of a colloid cancer of the rectum. In my sections there were certain areas in which the mucus-secreting elements did not predominate, but in which the cell arrangement was that of the young transitional epithelium, typical of that in the urachal canal.

Some Remarks on the Nature of these Colloid Cancers. — While the general appearances of the tumours bear a strong resemblance to rectal cancers of a similar type, there has been abundant opportunity to prove by operative examination and autopsy that they are primary, and indeed a very close investigation will reveal certain points of difference from intestinal growths and betray the true origin of the apical neoplasms. Some of these features have already been indicated.

"Rectal cancers as a rule produce a more developed and permanent type of granular cell which holds its form longer and is more stable than that in urachal tumours" (de Korte). In the latter, so great is the production of mucus, and so rapid the breakdown of the cell, that the formed elements occur over a very limited area. The cell goes through its furious cycle of mucin production and is soon destroyed by its own activity. So copious a production of mucin also catches, unawares as it were, many cells before they can undergo the process of metaplasia. The pressure of the enormous flood which finds no outlet 'fixes' on the walls of some of the cysts many rows of cells still in their primitive cubical or flattened form. They are crushed out of existence before they can take part in the fantastic riot and vicious activity of their fellows. Hence cubical or flattened epithelium still maintaining its primitive transitional formation is visible for a short period before it too becomes disintegrated in the general destruction.

Again, the metaplasic impulse is so violent that the topmost layers of cells become cylindrical and secrete mucus before going through the formality of arranging themselves in single file like well-ordered mucus-secreting cells. They exercise to the top of their bent their potencies, long held in reserve in the cellular lumber-room of the urachal lumen, while still in position as the topmost layer of the transition epithelium, and seem to endeavour by their frenzied activity to make up in a few days for the enforced inactivity of years.

The complete absence of a membrana propria has already been mentioned. De Korte notes the great thickness of the walls of the cells, as if they had undergone a process of hyperplasia.

CLINICAL HISTORY, DIAGNOSIS, TREATMENT, AND PROGNOSIS.

Before passing on to the rather more interesting subject of the etiology of the colloid urachal cancers, it is necessary to devote some time to the more practical aspects, in order to leave the field clear for the detailed study of the urachal cells and the transitional tumours, simple and malignant, which form stages on the way to the fully developed colloid cancer, or, falling short of this goal, branch into the less regular types, classified in this paper as ' mixed tumours '.

Clinical History and Symptomatology.—The clinical history differs from that encountered in vesical neoplasms in that certain symptoms due to the growth of the tumour usually occur before bladder trouble proper ensues; and indeed in one or two instances the growth was removed before it had actually penetrated the mucosa, and there was no disturbance of micturition. The exceptions to this rule are the small group which are malignant from the beginning, and penetrate into the bladder before they have had time to extend upwards. A number of tumours about the region of the vesical apex have been found at post-mortem examination. These had not caused the patients any trouble during life, and it can be well imagined that a growth must attain considerable size in the free space above the bladder before it gives rise to any symptoms at all. Occasionally by interference with the action of the urachus, which plays a by no means negligible rôle in bladder dynamics, there is some hesitation in starting the stream, mild frequency, and other bladder dysfunction—mere annoyances not sufficient in themselves to take the patient to his doctor. In Pendl's case, for example, in which an enormous tumour the size of an adult head was found, with a capsule in places 2 cm. thick, and composed of old sclerosed and avascular fibrous tissue with areas of calcareous deposit, the patient made no complaint until two months before examination, although the growth had probably existed for years.

In Green's case there was some frequency of micturition *fourteen years before*, which then subsided, and the patient was conscious of little disturbance until he himself felt the orange-sized tumour one year before he was examined.

The evidence seems to prove that at first the growth is non-malignant and produces no symptoms until it becomes very large, unless it breaks through into the bladder.

The first complaint, as a rule, is abdominal discomfort, sometimes itching, and tenderness when the patient presses his own abdomen—and at times

discovers the presence of a lump in the process. Often the first symptom is hæmaturia, and this, in most cases though not all, is followed by the inflammatory phenomena of pain on micturition and marked frequency. Later, pieces of tumour may be found in the urine. The inflammatory phenomena may mask the slight hæmaturia, as, in common with all other cases of malignant tumour involving the bladder, infection is apt to set in early.

On examination an abdominal swelling may be seen or felt, movable in its upper part, fixed in its lower. Dullness on percussion may be elicited, and, more commonly, even where no tumour is palpable, there is tenderness on deep pressure in the suprapubic region. Bimanual examination by rectum or vagina may elicit a tumour and locate its site as being high up in the bladder, or there may be intense tenderness during the examination.

Cystoscopic examination, to be fruitful, requires that the examiner be aware of the existence of such growths. They may be present merely as swellings or protrusions of the bladder apex, giving the impression that something is pressing from the outside. There may be merely what appears to be a flat epithelioma, though the circular form and the presence of normal bladder mucosa up to the very circumference of the lesion are marked features. In these cases there is usually a raised edge, and a small piece removed for examination will show the typical structure. In others there may be a funnel-shaped retraction of the bladder apex with a tumour ring around it or a tongue of growth emerging from its recesses. In still others the appearance may be of a localized tuft of polypi or papillomatous fronds projecting into the bladder. In Green's case there was a small hole such as may be seen in cases of normal urachus, but gelatinous fluid was being discharged from it. Wherever possible a small piece should be removed for examination so that the error of treatment by fulguration or by radium may be avoided.

Differential Diagnosis. — The growth is to be distinguished from the common types of tumour found in other parts of the bladder wall. The rarity of these at this site, however, demands a thorough clinical examination for the presence of a suprapubic tumour, and the removal of tissue for study. If the tumour is palpable above the symphysis, the differential diagnosis from neoplasms of the omphalo-mesenteric duct, echinococcal cysts, abscesses in the space of Retzius of urachal or other origin, desmoid tumours, lipomata, and cysts arising within the abdominal cavity, has to be considered. This presents little difficulty if the possibility of the occurrence of the urachal tumour is kept in mind. Early adenomatous formations arising from the urachus must be distinguished from endometriomata, whose structure they may closely simulate.

Age Incidence (by decades).—

Years	Cases
20–30	1
30–40	2
40–50	7
50–60	3
60–70	4
70–80	0
80–90	1
Total	18

No case younger than 29 has been recorded, and the larger number of the patients were over 40. Examination of the urachus from routine specimens shows that the small adenomatous formations do not develop to any extent until middle age. In this respect there is a correspondence to Lendorf's observation that the glandular structures in the bladder develop very slightly till after puberty, and are much more frequent and widespread after middle life.

Sex Incidence.—Of the 18 cases, 14 occurred in males and only 4 in females, showing a decided preference for the male sex. The significance of this feature is not clear, but it is difficult to associate with the richer development of prostatic glandular tissue in the male.

Treatment.—The treatment is based on the knowledge that metastases are rare, but that since they are very common after interference the operation must be radical, and based on the consideration that one is dealing with a tumour in the urachus whose connections, direct and indirect, may reach in abnormal cases as far as the umbilicus. Metastases after operation occur not uncommonly in the space of Retzius. The tissue removed must include the umbilicus with an area of at least half an inch of skin round this, a wedge-shaped block including the transversalis fascia and peritoneum wide of the growth, and the upper part of the bladder. In deciding on the amount of bladder to be removed, consideration should not be given merely to the area of mucosal involvement, because the part of the muscular wall involved is always greater in diameter (*see Fig.* 325). At least the upper half of the bladder should be removed. In the past there has been an attempt, always doomed to failure, to preserve the peritoneum. The opposite principle should be adopted, as much of this structure as feasible from the point of view of securing apposition being removed with the growth.

Prognosis.—No evidence is available as to the malignancy of these tumours if left alone. As they resemble colloid cancer of the bowel in the fact of the mucin being secreted by the cells, rather than the relatively benign cancers of the breast* where it may be in part due to degeneration of the stroma, one would expect them to be very malignant.

The long history in most cases indicates rather that the cancer has superimposed itself on a non-malignant tumour than that it is in itself of a relatively benign type. The evidence on this question, however, is incomplete. There is more information available in regard to the progress after operation. My case died two and a half years later from metastases without bladder recurrence. No report earlier than this can be safely considered as an index of cure.

Not one of the 18 cases of the series which was followed up for more than two and a half years was alive at the end of that time with the single exception of Lavenant's patient, who was apparently well but had had a hemiplegic attack, the latter being in itself significant from the point of view of metastases. On the other hand, the immediate operative results were good, and in

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* My acceptance of the traditional view that colloid cancers of the breast are relatively benign has been modified since writing the above after a conversation with Dr. Broders of the Mayo Clinic. In the abundant material at his disposal Dr. Broders has found that 90 per cent of such cancers must be graded as 3 on his scale, and that in the formation of metastases and resistance to treatment they are on the whole more malignant than non-colloid carcinoma.

many cases there had been no recurrence in the bladder after one year. Metastases elsewhere do not as a rule declare themselves in that time. This seems to show that the growths are of only average malignancy.

Secondary neoplasms of the bladder derived from other organs through the blood-stream are very rare; secondary colloid cancers of the type under discussion have not as yet been recorded. A few clinical cases are reported in which metastases of the bladder apical tumour were widespread. In these the growth was found at atopsy. De Korte's case probably had a large mucinous mass in the pelvis before operation, but in none of the others was there any reason to believe that metastases were present before operation. They occurred afterwards in the abdominal wall and the bladder itself, but it must be remembered that the operations planned did not conform to the radical one I have suggested.

A SURVEY OF THE REMAINING CLASSES OF ADENOMATOUS AND 'MIXED' TUMOURS ARISING FROM THE URACHUS AND INTIMATELY INVOLVING THE BLADDER.

The clinical study of the colloid adenocarcinomata of the bladder vault has now been completed. Before proceeding to an examination of that chain of events by which the primitive cloacal cells of the fœtus become the active malignant ones of the adult, it will be as well to consider shortly in some of their clinical and histological details the two groups which have been referred to as the adenocarcinomata and the 'mixed tumours' (*see* p. 434). Following that, a brief reference will be advisable to the group of mesodermal growths, which in my opinion arise from extra-urachal structures. These have, of course, no bearing on the etiology of colloid cancers, but as they have been described again and again as tumours of the urachus, they cannot be altogether ignored in a study concerned with that structure. There remains the group of urachal rests (*Class IV*, p. 434). Finally, a brief reference will be made to the adenomata of the umbilicus, in order to illustrate the similarity in form which growths derived from the fœtal hind-gut assume when they take on neoplastic activity in the adult.

Adenocarcinomata without Colloid Degeneration.—It is a striking fact that so few examples of simple or non-colloid adenocarcinoma have been reported —only three in all (*see* (*d*) and (*e*) in table on p. 434).

It is remarkable that while in the case of the colloid growths there is reason to believe that the results were uniformly bad, two of the three cases of adenocarcinoma were well after nine and ten years respectively, one of them after having had 12 in. of intestine removed with the tumour.

In some instances of colloid formation—illustrated, for example, in the reports of Barringer and Pendl—the cells had taken on active secretory activity while still cubical. Taking this into relationship with the paucity of cases of simple adenocarcinoma, one is led to expect that the phase of columnar-celled formation is practically synchronous with mucin secretion and malignancy. The urachal cells hasten feverishly to begin the activity which as entodermal cells has long been denied them, not even waiting to assume the regulation cylindrical shape before starting their secretory riot. In the three

cases of adenocarcinoma, where they had become frankly columnar and still exhibited but little energy, we are doubtless faced with an exceptional instance of unusual tameness and docility on the part of the epithelium.

'Mixed Tumours'.—The term 'mixed tumour' in this connection is somewhat of a *petitio principii*, as it is doubtful if the neoplasms so described were really of the nature of teratomata. The group, however, present unusual and diverse pathological features, and more than one author considered that his case was an example of a true teratoid tumour. Ewing classified Barringer's specimen as such, though it seems to fall in line with the other colloid adenocarcinomata. The reports of the cases here referred to (*see* (*g*) and (*h*) in the table on p. 434) all concern neoplasms closely connected with the bladder apex. They were considered by those who reported them to be derived from urachal or 'allantois' remnants. Some of them present features which remind one very forcibly of certain formations found in connection with the urachus, and it is possible that they represent an unusual malignant development of the microscopic adenomata. Similar types are not unknown arising from the bladder mucosa proper (*see* p. 437).

Some of the tumours which contain sarcomatous and epithelial elements side by side are probably examples of growths in which both the mesodermal sheath and the epithelial canal take part. In some cases the derivatives of the former predominate, giving a picture of a sarcoma with a few glandular spaces. The competition of the mesodermal section modifies the form of the acini, but the cubical cells, sometimes of more than one layer, which line the spaces and the papillary projections are reminiscent of certain urachal appearances.

Mesodermal Growths Arising from the Muscular and Fibrous Tissue of the Urachus and from the Ligaments connecting the Urachus with the Umbilicus.—All the ten cases (*see* (*m*) and (*n*) in the table on p. 434) were of the same type. They did not arise near the bladder, and it is probable that all developed from the strands or ligaments connecting the urachus with the umbilicus. They have no bearing on the subject under discussion, except that they are described and reported as urachal tumours. Most of them occurred in females, in contradistinction to the true urachal tumour, which as a rule is found in adult males.

Reference has now been made to all reported bladder apex tumours which appeared to originate in the muscular wall, as far as I have been able to find them in the literature. In addition, all growths which have been thought by the authors to be of urachal origin have been commented on, with the exception of the cases of Nicholson, Hector, and Hagner, which were simple fibro-adenomata discovered at autopsy or exploratory operation and will be discussed later, and those of Koslowski, Adám, and Hue and Jacquin, which are successive stages of neoplasms developed from urachal rests above the adult position of the urachus itself. These also will be considered in a later section.

There has been no selection or elimination of cases to prove a thesis. The striking fact emerges that the thirty-one tumours arising from the bladder apex were either colloid adenocarcinomata, adenocarcinomata, fibro-adenomata,

or 'mixed tumours' mostly having a very definite glandular aspect. The cases of Doiran and Cullen cannot be definitely classified, but they were unusual growths of uncertain nature. On the other hand, the ten tumours which arose from mesodermal tissue all developed above the bladder, and involved the latter, if at all, only by direct spread along the abdominal wall.

This is a striking confirmation of the anatomical findings that the urachus with its epithelial canal extends only 5 to 8 cm. from the bladder apex. It also seems to justify the statement that true tumours of the urachus always have an epithelial element, and that those which do not satisfy this criterion should be discarded from the category.

PART II.

THE ORIGIN AND HISTORY OF THE EPITHELIUM OF THE URACHUS, AND THE PATHOLOGICAL CHANGES IN IT WHICH LEAD TO THE DEVELOPMENT OF THE COLLOID ADENOCARCINOMATA.

The primitive hind-gut in the early fœtus is the anlage of the rectum, the upper part of the bladder, and the urachus. This cavity is, according to Wesson,[130] lined by a single layer of cuboidal cells. A tendency for these cells to become more cylindrical in shape on the one hand, and to form by their subdivision two superimposed layers on the other, may be observed very early, adumbrating the tendencies which are finally brought to fruition in the formation respectively of the cylindrical mucosa of the rectum and the transitional epithelium of the bladder. The complete differentiation of the types takes place only after the interposition of the urorectal septum has determined the limits of the two cavities. Equal potencies are inherent in all the cloacal cells, and the ultimate form which they will assume is determined only by the physiological rôle assigned to them. The exact point of subdivision effected by the urorectal septum is arbitrary to this extent, that it does not separate a group of cells predestined to form rectal mucosa from another group equally predestined to form transitional epithelium, following as it were a trail already blazed where two histological continents march side by side, or building a wall along an international frontier previously determined. Such exactitude is at variance with our experience of embryological actualities. The further development of the individual cells is decided by the position which the septum takes up rather than the latter by any inherent difference in the nature of the cells.

While this view is probably so universal that its expression is almost a platitude, the theory of rests or abnormally persistent rectal epithelium has much acceptance, but appears to be entirely illogical. Misplaced intestinal mucosa has been invoked to explain colloid cancers occurring in the bladder area. The only assumption on which such a proposition can rest is that certain areas of the epithelium of the cloaca have limited potencies compelling them if they persist to become rectal mucosa in the case of certain cells on the one hand, or transitional epithelium in that of others—the two groups being fundamentally distinct. After the urorectal septum has formed, the cells of the former group which find themselves anterior to the partition are said

to die out, as they have no power to become transitional epithelium, and find themselves accordingly in an anomalous position. If they persist they remain as 'rests' of rectal mucosa and may give rise to appropriate types of tumour.

If this theory were correct, one would expect to find intestinal growths in the bladder as the rule rather than the exception. The proposed axiom of the death of cells which find themselves in a teleologically unsuitable situation is hardly less fantastic than the supposition that the urorectal septum will neatly divide the narrow line between two individual cells of an entirely different nature.

The whole theory of vestigial rests has been worn threadbare to explain difficult pathological phenomena. The only logical point of view is to think of primitive cells* such as exist in the early cloaca as being totipotent as regards the formation of neoplasms ectodermal, entodermal, or mesodermal. The assumption of the specificity of the three primary germinal layers belongs to an older pathology. The theory certainly had its attractions, for by juggling with this trinity of neoplastic ancestors and using the trump card of vestigial rests to retrieve a losing trick, a more or less plausible explanation could be given of any and every pathological phenomenon! When, however, we meet with squamous epithelioma of the gall-bladder, uterus, bladder, and kidney pelvis, carcinoma of the mesodermal renal tubules, and similar phenomena, our credulity in this axiomatic specificity is stretched to the breaking-point and inevitably compels a belief in a higher potency latent in body cells than the older pathological opinion would admit.

While multipotency, if not totipotency, must be admitted for all cells even up to an advanced period in their life-history, there remains a strong tendency for the three primary layers, ectoderm, mesoderm, and entoderm, to develop along their own special lines. Any idea, however, of their absolute specificity ought to be discarded, and metaplasia looked on as a phenomenon not excessively rare.

Returning to the primitive cloaca, we find that the cells posterior to the urorectal septum develop into the highly organized structures of the intestinal mucosa with its glands, its stately columnar epithelium, and its mucous cells; those anterior into the well-morticed architecture of vesical epithelium. Hardly have these baby cells of the hind-gut begun to develop, however, than a further subdivision takes place—a partitioning of a very different type from the former, which reproduced in ontogenetic miniature the phylogenetic emancipation of the higher genera from the disadvantages of the rather too universally applicable cloaca! This second subdivision, although humbler in its object, is not without purpose. By it the upper part of the bladder is narrowed down as a prelude to the descent of that viscus behind the safe shelter of the symphysis pubis. This migration has a double aim. It guarantees the organ

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* The limit of age beyond which adult epithelium becomes incapable of metaplasic change to another type—if indeed any limit exists—is undetermined. That totipotency as a quality is not confined to primitive cells is evidenced by the formation of ectodermal neoplasms from developed entodermal varieties, as seen in the squamous-celled epitheliomata of the gall-bladder as well as the urinary bladder. There is probably a reversion to a more elementary type of epithelium as a prelude to the metaplasia (*see* p. 450 and *Fig.* 339).

against injury, to which its fixed position outside the peritoneum and well to the front of the abdominal wall would expose it, and at the same time removes its apex from the weak point of the umbilicus, rendering more secure this final point of closure of the fœtal leaf, and removing the danger of urinary fistula involved in such intimate association between a frequently distended viscus and a vulnerable point in the abdominal wall.

It is not advisable, however, that the bladder should lose entirely as its *point d'appui* such a convenient point as the umbilicus, since the vesical apex must be steadied to fulfil satisfactorily the function of micturition. The formation of the musculo-tendinous apparatus of the urachus and the ligamentum commune fulfils all requirements (*see Fig.* 328).

The ideal is, however, not absolutely attained, for the epithelial canal left in the urachus has no possible place in the physiological scheme. The epithelia of the bowel and bladder fulfil their respective destinies to meet the needs of the organism; that of the urachus has no destiny at all. It is merely flotsam and jetsam, builder's refuse thrust out of the way among the joists. But though its *motif* is lost, its vital impulses remain. It does not die, and yet it has no useful purpose in living. It remains fixed at that stage of development which it had reached at its segregation, primitive epithelium. There is no imbrication, morticing, and welding of the cells with each other—the mutual adaptation by which their more fortunate brethren co-operate to form the perfect waterproof lining of the bladder. The urachal cells remain individualists. They heap themselves up on each other without order or regularity (*see Figs.* 334, 335). That the masses of embryonic elements form a complete lining to the urachal canal many layers in depth is an accident of reproductive activity rather than the end-result of any teleological aim. These pariahs and outcasts from the corporal community exhibit the unbridled procreative powers of the unfit. Primitive cell multiplies from primitive cell in the narrow prison house. Reproduction and degeneration, degeneration and reproduction, follow one another in unending cycle. The cells jostle and destroy their fellows in the struggle for room in which to exist. Individuals perish, and the débris from their remains fills the narrow urachal canal from which there is no escape. The force exhibited in cellular reproduction is immense. By its means the tender weed forces its way through solid asphalt, and roots wedge rocks apart. In the urachus the new cells formed at the periphery, finding no space in the crowded lumen, press out into the sheath; and in that narrow and unguarded area where the tube, devoid of any fibrous protection, passes through the muscular bladder wall to reach the mucosa, the expansion and contraction incidental to the act of micturition alternately release and compress the cellular column and give still further impetus to the unbridled behaviour of its inmates. In this way the scene is set for the formation of these irregular cell-growths which predominate in later life, but which fortunately progress as a rule too slowly to become a menace before death ensues from other causes.

The retention by the urachal cells of all the potentialities of primitive cloacal cells, and their ability to bring them at any period of life to untimely fruition, are the keys to the understanding of the various types of tumour of the urachus. Complete but ill-regulated development towards glandular

or transitional epithelium respectively may be observed, may overstep all bounds, and give rise to colloid adenocarcinoma on the one hand or squamous-celled epithelioma on the other. Both these types were seen in a single tumour in the sixth case of Rankin and Parker. As a rule, however, the full impulse given by the entodermal origin of the cells predominates, and the formations commonly seen pass through the stages of simple adenoma and adenocarcinoma to reach finally the fully formed colloid cancer of the rectal type. It is fortunate that this transformation starts as a rule too late or progresses too slowly to reach the threshold of clinical observation during the normal span of life, but it may commonly be observed by microscopic study of the urachus in routine autopsy specimens. Sufficient points of resemblance exist between these microscopic tumours and those of clinical importance to enable them to be identified as different stages of the same process.

RELATION OF URACHAL ADENOMATA, ETC., TO THOSE ARISING FROM THE PROPER EPITHELIUM OF THE BLADDER.

What has been stated in regard to urachal epithelium applies also in a modified sense to that of the bladder itself. The cells of the latter are of the same origin and theoretically retain the same latent potencies. In point of fact, adenomata, adenocarcinomata, and colloid tumours of the rectal type *do* occur in the bladder, and have been proved to arise from the epithelium of that organ. The transitional epithelium may become converted into cubical, the latter into cylindrical, and finally adenocarcinomata of the intestinal type, with or without the excessive secretion of mucin, may result (*see Fig.* 339). The primitive urachal cells, however, which retain their early cloacal characteristics, may develop along the divergent paths which lead towards vesical or rectal epithelium with equal facility. On the other hand, those of the bladder have already advanced a considerable distance along one of these diverging paths, and to be converted into the rectal type must either retrace their path to the starting-point and then proceed by this circuitous route to the goal of intestinal mucosa, or, alternatively, make the incredibly difficult metaplasic leap over the gulf which separates the transitional from the rectal type. Considering the enormously greater quantity of epithelium in the bladder compared with that in the urachus, these changes are correspondingly much rarer in the former organ than in the latter.

MICROSCOPIC ADENOMATA AND CYSTS OF THE URACHUS, AND THEIR FORMATION.

Anatomical Considerations. — We know that the urachal canal is surrounded by a stout fibromuscular sheath as far as where it pierces the superior bladder wall, and that below this point it passes to the mucosa without any such protection, lying free in the muscular coat of the bladder. In conformity with this anatomical fact it has always been found that malignant tumours are fairly well encapsulated above, but invade the tissues unhindered in the intramural portion.

If a number of specimens of the urachus are taken at random, it will be found that the canal can be easily dissected out from its sheath, which hides

the irregularities in its contour ; and it will then be found in a large percentage of cases to exhibit nodulations and tiny cystic formations, more especially in those parts that lie immediately above the bladder and actually in the vesical wall (*see Fig.* 337). Where the lumen fails to maintain connection with the bladder cavity, the blind end at the level of the external surface of the bladder mucosa is frequently dilated to form a bulbous swelling microscopically similar to the cysts projecting from the lateral wall (*see Fig.* 338).

Luschka[80] observed diverticula and cystic formations in the urachal canal in most of the specimens examined. In the 74 cases of Wutz[132] no fewer than 24 exhibited cystic dilatations. In a majority of the ones I have examined

FIG. 340.—Section taken through the muscular wall of the bladder apex. There are two urachal canals. One has been cut partially in longitudinal section.

there were either definite projections as large as barley grains visible to the naked eye, or microscopical examination revealed multiple canals parallel to the main one (*Figs.* 340, 348), columns of cells lying free (*see Fig.* 346), small cysts lined by cubical or flattened epithelium (*see Fig.* 343), or complex structures having the appearance of fibro-adenomata (*see Fig.* 344). The cysts appear in all cases to be derived from degeneration of the central portion of solid columns or masses of cells in the first instance, with successive multiplication of the limiting cells and degeneration of the more internal ones, until comparatively large cavities were formed (*see Figs* 341, 342). It seemed very questionable if any of the contents of these *early* cysts owed their origin to

actual secretion from the lining cells. They were true cystic adenomata, and not due to retention of secretion.

These microscopic structures have probably no relation at all to the enormous cysts appearing in the literature under the caption of urachal cysts. The latter owe their origin to pathological processes of various kinds, mostly having little or no connection with the urachus. The small adenomata and cysts found on examination of routine specimens have, however, their counterpart in larger structures, such as the specimens reported on by Nicholson[87] and Hagner.[49] A more advanced stage may be seen in a specimen of a spindle-shaped cyst $2\frac{1}{2} \times 1$ cm. in cross-section in the Museum of University College Hospital, London. It was found incidentally during an operation on a woman for some pelvic condition.

Changes in the Clinically Normal Urachus.—Some examples of the various changes to be found in the clinically normal urachus will now be described in detail and illustrated by specimens selected to demonstrate the different phases, beginning with the earliest and passing on to the more complex.

The following is in general the sequence: The cells of the urachal canal are constantly proliferating, by the amitotic method. The effect of this is twofold. On the one hand, they become heaped up towards the lumen, some being desquamated into it (*see Fig.* 334); on the other, the peripheral cells extend in a centrifugal direction and are cut off from the main column by the ingrowth of the fibrous tissue of the sheath (*see Fig.* 341). The separated packets continue to multiply, forming masses, but more generally extending as solid columns for some distance parallel to the direction of the urachal canal. By the degeneration of the middle cells, presumably from insufficient blood-supply, a lumen is formed. A cross-section at this stage will exhibit the large central urachal canal flanked by other smaller ones, all lined by several layers of cells (*see Fig.* 340, 346). When the separated groups assume the form of a mass rather than a column, the extension by multiplication of the peripheral units, together with the successive degeneration of those situated towards the centre, leads to the formation of a cyst filled with amorphous content and the remains of cell membranes and nuclei (*see Figs.* 345, 347). The increase in circumference of such a cyst produces a thinning out of the epithelial lining, and the pressure of the contents a flattening of the lining cells. There may be only one or two layers of the latter.

If numerous masses and columns of cells are cut off rapidly, the neoplastic appearance of an adenoma is formed in which both solid columns and cystic structures appear side by side, and the cells in such cases may change from their primitive form and become elongated, cylindrical, and finally actively secreting cells. At this point the tumour has definite malignant tendencies.

Another feature observed is where the fibrous stroma pushes into a cystic cavity in a tongue-shaped manner carrying the epithelial layers with it and forming a papillary projection (*see Fig.* 350). The fibrous core of such a papilla is covered with the layers of the epithelium originally lining the walls of the cyst which it has pushed in front of it when it invaded the cavity. These papillary formations are a prominent feature of some of the 'mixed tumours'.

Specimen 1.—**Illustrating the Formation of a Urachal Cyst** (*Figs.* 337, 341, 342).—The urachus was taken from the body of an old man undergoing dissection. No clinical history was available. The specimen had the appearance of a normal urachus and was 5 cm. in length. The canal extended to a point 4 cm. above the bladder. On removing the fibrous sheath two small smooth cystic projections

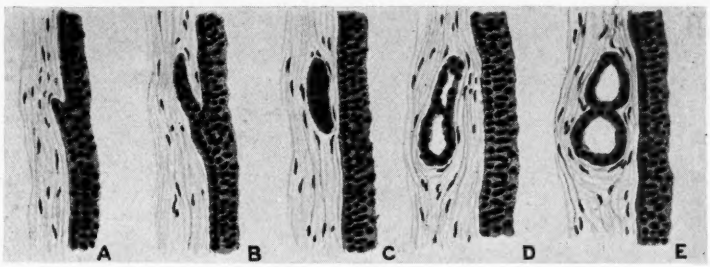

FIG. 341.—The beginnings of a cyst. Sections taken from above down in specimen shown in *Fig.* 337. A, Epithelial bud into the fibrous tissue; B, A mass of cells formed still attached; C, The mass is separated from the parent epithelium; D, The central cells have degenerated and subdivision is commencing; E, Subdivision is complete.

were seen projecting laterally from the outer wall of the structure at a point 1 cm. above the bladder mucosa (*see Fig.* 337). They were sessile, 2 mm. in diameter, and their thin capsule was formed by an expansion of the fibrous coat of the canal.

MICROSCOPICAL EXAMINATION.—Serial sections were cut from the upper level of the urachus to the bladder, including the cysts.

The canal was of normal appearance till a point was reached 1 mm. above the level of the upper cyst (*see Fig.* 337). Here the outermost cells of the epithelial lining formed a little group of three or four budding into the connective tissue (*Fig.* 341 A and B). A little lower the group of cells was larger, and was separated from the canal by young fibrous tissue (*Fig.* 341 C). Still lower the central individuals were breaking down (*Fig.* 341 D), thus forming a lumen surrounded by several layers of cells. The small cyst thus formed was continuous with that microscopically visible below. It had divided into two, but the cavities as seen in the section had again connected with each other and with the lumen of the urachal canal (*Figs.* 341 E and 342). An accumulation of cells marks the point of amalgamation of the two epithelia, that of the cyst and that of the urachal lumen. Both lumina contain amorphous content which has shrunk away from the walls in the process of hardening.

The steps in the formation of this cyst may be summarized as follows:—

1. Bud of cells pushing out into the fibrous membrane lining the canal.

2. The multiplication of these cells, forming a mass separated by young fibrous tissue from the canal proper.

3. The breaking-down of the central cells forming a lumen, and the division of the hollow columns into two.

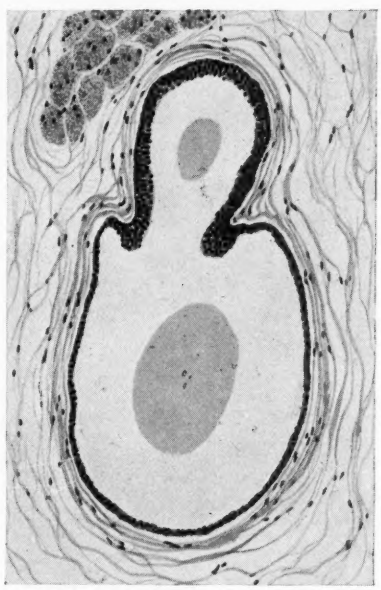

FIG. 342.—A section through the upper cyst shown in *Fig.* 337. The new cyst has again joined the urachal canal, which shows some dilatation. Note the thicker epithelium of the cyst, the amorphous substance filling the cavities, and the degenerated nuclei in the canal. The cyst content has shrunk in the process of hardening.

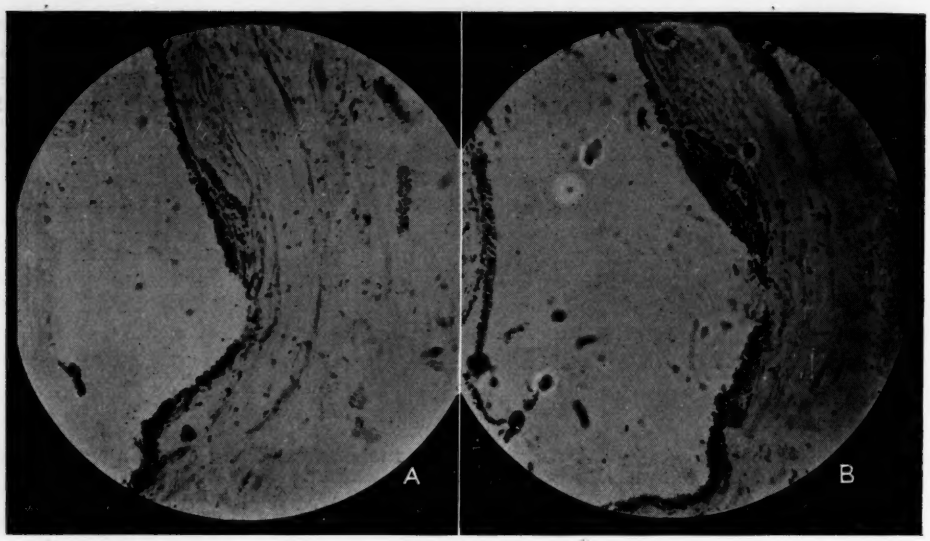

FIG. 343.—The upper and lower of a group of serial sections taken from a small cyst at the lower end of a urachus. In A a group of cells is isolating itself from the urachal epithelium. In B two small cysts have formed from this group lateral to the main cyst wall.

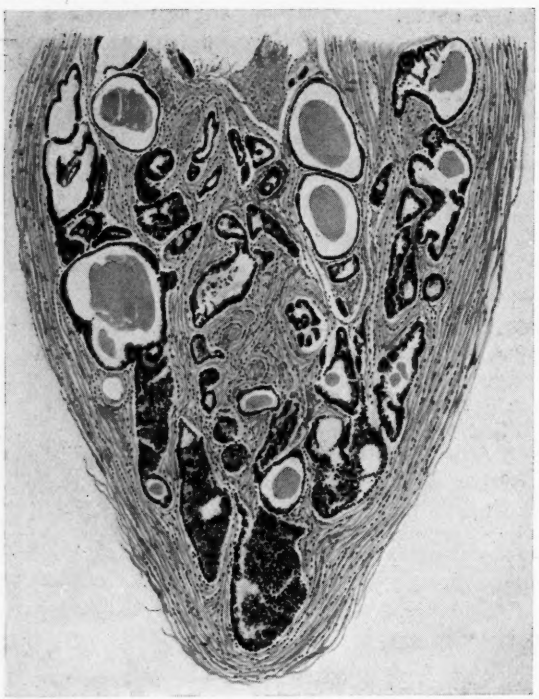

FIG. 344.—Section of the lower part of the supravesical urachus from a woman of 47 years. The illustration represents half the total area of the urachus. There is adenomatous formation, and the growth has invaded the fibrous sheath.

4. The gradual increase of these separate cysts till their cavities have again burst into each other and into the urachal canal.

Lower down, the urachal lumen and the cyst which had burst into it became one homogeneous whole, so that the section through the lower cyst shown in *Fig.* 337 presented the appearance of a wide dilatation of the urachal canal. The lining epithelium consisted of only two layers of very flattened cells.

The tendency to subdivision either directly or after the lumen has again filled up with cells is illustrated in *Figs.* 342 and 343. The specimens were from two separate cases.

Specimen 2.—**Illustrating the Formation of an Adenoma of the Urachus** (*Figs.* 344–348).—The specimen was removed from the body of a woman of 47 who had died following the operation of hysterectomy. The urachus was 6 cm. in length and had a normal appearance. A section made just above the bladder illustrates a latent type of adenomatous formation (*Fig.* 344).

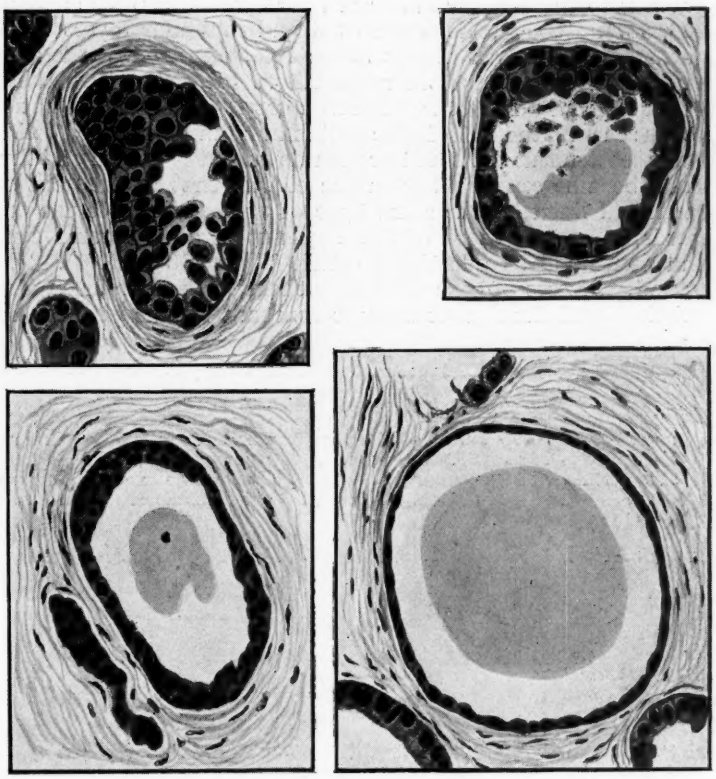

FIG. 345.—A series of high-power views of the same section as that shown in *Fig.* 344, showing how a round cavity lined by flat single-layered epithelium is formed by the degeneration of the central part of a column of cells. The débris resulting from this process filled the lumen, but retracted during the hardening of the tissue.

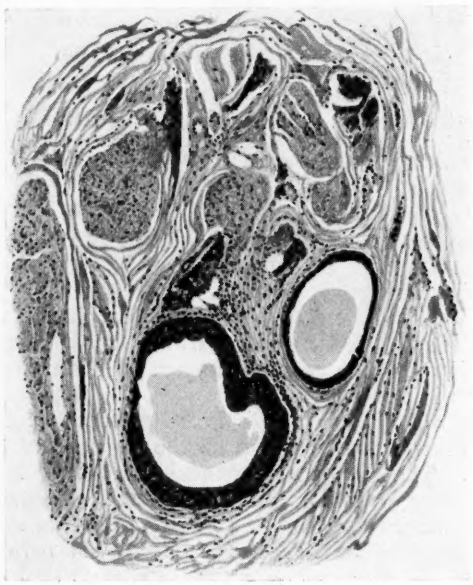

FIG. 346.—Section taken from the upper part of the same urachus as shown in *Fig.* 345. Note the two canals, the difference in the number of layers of epithelium, and the mass of detached cells above the canal on the left.

MICROSCOPICAL EXAMINATION.—Serial sections from the upper end of the urachus to the bladder illustrated the formation of the adenomatous structure (*Figs.* 346, 347). Above, the adventitial and muscular layers of the urachal sheath were normal. The lumen was at first single, but soon divided into two separate canals, each lined by the customary primitive transitional epithelium (*Fig.* 346). At the upper pole of one of these canals a group of cells could be seen pushing out into the fibrous membrane outside the epithelium, and forming a solid mass or column. In a lower section this mass had broken down in the centre and formed a lumen. The new canal became separated from the old, so that in the next section there were three canals all lined by the same type of epithelium. As the sections proceeded downwards, more and more of these groups of cells became separated, formed lumina, and continued as additional canals (*Figs.* 348 A, B, C). The process became widespread. Masses of cells detaching themselves came into contact with others already existing, and the figures became confused by the junction of two epithelial masses,

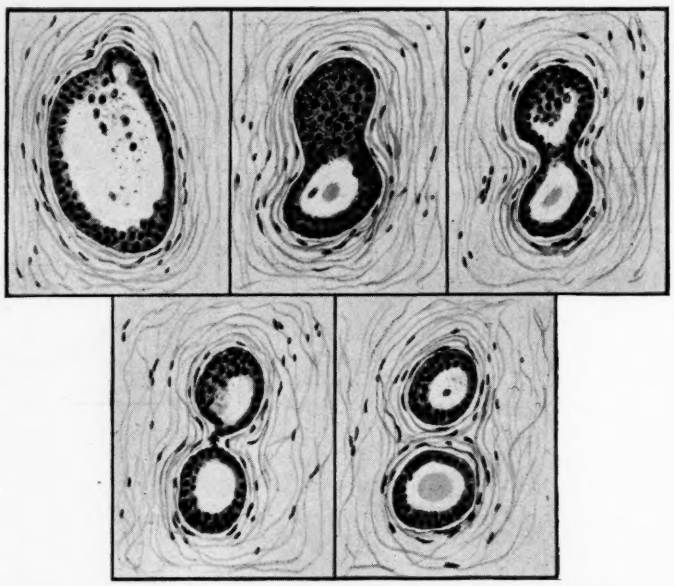

FIG. 347.—From the same case as that shown in *Fig.* 346. Thick serial sections from above down to illustrate the method of formation of two acini from one. Note the condensation of the fibrous tissue and the way in which it conforms to the subdivision of the acinus.

the amalgamation of two cysts to form one cavity, or the subdivision of one to form two. The section now began to have the appearance of an adenomatous tumour, and spread outside the fibrous membrane of the urachal canal proper, invading the muscular and fibrous sheath (*Fig.* 344). Towards the bladder below the sheath the acini, if they can be so called, maintained an orderly arrangement, did not penetrate between the muscle fibres, and stopped short at the external aspect of the mucous membrane—the lower limit of the urachus in this case.

Fig. 344 shows the structure at the widest point. Its boundaries are coterminous with those of the urachus. The adenoma has grown at the expense of the muscular sheath without expanding it. The outer adventitial coat has been thinned out, but still exists as a fairly regular fibrous capsule indented in places by the pressure of cysts nearest to it. The stroma consists of young fibrous tissue. All formations are seen, from irregular masses of epithelial cells having the general character of urachal cells, to cystic spaces. Most of the latter are surrounded by a condensation of the fibrous tissue. Numbers of them are scattered through the

stroma. The cavities contain an amorphous content which does not give the reaction of colloid (*Figs.* 344, 345).

The epithelial lining of the cysts varies. In some, apparently the more recent, the many layers of cubical epithelium similar to normal urachal epithelium are seen, while in other parts of the same cyst there is a single layer of cubical or flattened

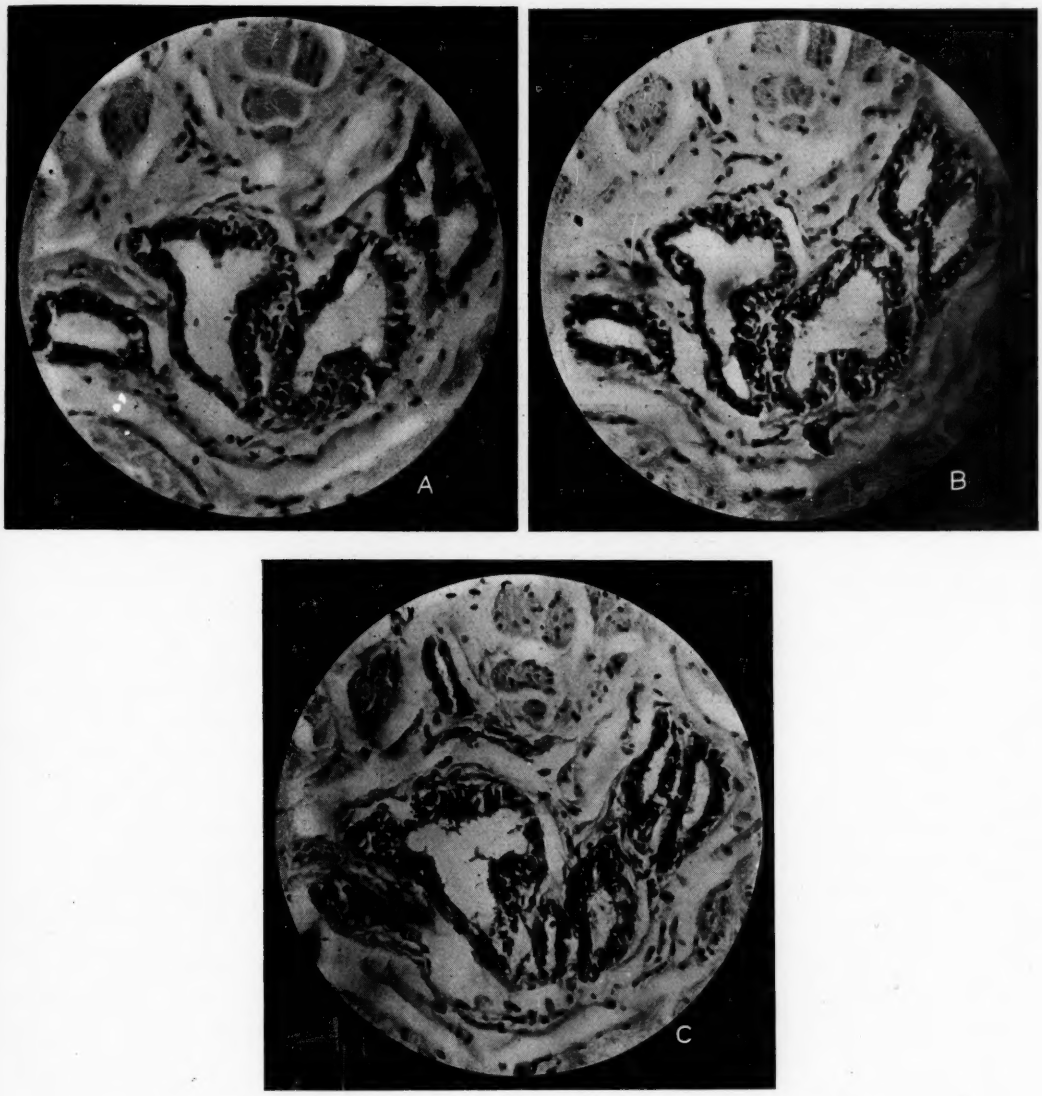

FIG. 348.—Three sections of the same urachus as shown in *Figs.* 344–347 taken some distance apart to show multiplication of the number of canals, a process which finally produces the type of structure seen in *Fig.* 344. Two single canals in A have subdivided completely in C.

cells. In a few of the spaces where the cyst is not distended but has an acinar appearance the cells are becoming cylindrical, but there are no goblet forms.

A few of the larger cavities have a clover-leaf appearance with more layers of epithelium at the angles where the components join, suggesting that these have been formed by the amalgamation of two or more smaller cysts.

From the original urachal cells have been derived all the various types of epithelium seen—primitive, transitional, flat, all grades of cuboidal, and finally cylindrical. Similar varieties may be found in the developed adenocarcinomata. None of the cells have as yet started to secrete mucin, and there is no evidence of malignancy.

These specimens have been selected to illustrate the formation of a small cyst and adenoma respectively. The processes involved are essentially similar, and in the numerous cases where I have observed them exactly the same series of changes can be followed. The two selected are so typical, however, that nothing would be gained by multiplying examples. The process is always the same budding of epithelium, separation from the main canal, lumen formation through degeneration of the central cells, subdivision of cysts so produced, and finally the formation of spaces lined by cuboidal or flattened epithelium. In the later stages the tendency is for the cells to develop into cylindrical forms.

Goblet and mucous varieties were not seen in any of the specimens I examined, but Suchannek[118] reports them in two of his cases.

FIBRO-ADENOMATA OF THE URACHUS AT THE BLADDER END.

This group follows naturally, as the cases represent a more advanced stage of the formations that have just been described.

NICHOLSON'S case.[87]—

Male, age 47. The patient died of heart disease, and had had no symptoms referable to the growth. The latter was in the lower end of the urachus immediately above the apex of the bladder. It was 5·7 cm. in length from above downwards and 4 cm. in breadth antero-posteriorly. Thanks to the courtesy of Dr. Nicholson, I had the opportunity of examining the slides. The tumour had a fibrous capsule, and was occupied by a number of cysts and tubular spaces. The latter were lined by irregular columnar or cubical epithelium. Papillary projections from the stroma were seen in the glandular spaces. The author's illustration shows that these papillæ are covered with cells tending to become cylindrical in type. In certain parts there is more than one layer lining the cavities, which contain amorphous débris similar to that found in the smaller specimens already described.

HAGNER'S case.[49]—

Male, age 68. During an exploratory cystotomy a tumour was palpated projecting into the upper part of the bladder. It was submucous, and was attached to the apical region of the bladder. The growth was removed and sectioned.

EXAMINATION OF SPECIMEN.—The tissue consisted of glandular spaces in a fibrous stroma. There were 'inflammatory' processes in which the cells were degenerated.

Hagner gives no exact microscopical description of his specimen, but it appears to have been a fibro-adenoma of the same type as in Nicholson's case. The cells, however, were beginning to invade the stroma, and the tumour probably represented a transition stage between the simple fibro-adenomata and the adenocarcinomata.

Some years ago Dr. Hector, of Wellington, showed me a specimen of a bladder removed from the body of a man found dead. There was a spindle-shaped swelling the size of a walnut at the lower end of the urachus. The tumour was smooth and contained in an expansion of the urachal muscular sheath. Its lower end rested on the bladder. Microscopically it had an adenomatous appearance, but unfortunately the specimen and slides were

not preserved, and no detailed description was made at the time. In macroscopic appearance it was remarkably similar to the specimen shown to me by Dr. Nicholson.

As pointed out above, the clinical history of the majority of the cases of colloid cancer suggests that the malignant changes supervene in a simple tumour which has already been present for a considerable time. We can trace the steps in a fairly systematic manner. First of all the microscopic adenomata, then the larger types as seen in Nicholson's case, the same type showing evidence of early malignancy as in Hagner's, the adenocarcinomata with the beginning of mucin secretion in the cells as described by Hartmann,[50] and, finally, the fully formed colloid tumours.

In the case of the small and very malignant growths confined to the intramural urachus (cases of Kielleuthner, Albarran, de Korte, and that of the author), the intervening stages appear to be absent, the urachal cells becoming actively malignant from the first. The observations of Suchannek[118] are particularly interesting in this connection. He made a study of a series of specimens of the urachus, and not only confirmed the frequent occurrence of cystic and adenomatous formations, but found examples in which the cells lining the acini were columnar in type, and in two instances showed *goblet cells and mucin in the lumen.*

These two cases were apparently on the borderline of malignancy, and no doubt if the patients had lived longer would have become fully formed colloid cancer without the intermediate stage of a macroscopically visible adenoma of the simple type.

The small proportion of cases of colloid cancer with their distribution restricted to the wall of the bladder harmonizes with the rarity of the discovery of these columnar and goblet cells in the routine examination of the urachus. The epithelium in the vast majority of cases attains only to the cubical or low cylindrical stage, and if it goes on to tumour formation, the tumours will in the first instance be simple fibro-adenomata.

EPITHELIAL TUMOURS ARISING ABOVE BETWEEN THE URACHUS AND THE UMBILICUS FROM URACHAL RESTS.

The class of growth which arises above the normal limits of the urachus—that is, in the upper two-thirds of the interval between the umbilicus and the symphysis—have already been referred to (pp. 431, 432). Most of these are mesodermal neoplasms, fibromyomata, fibromata, and sarcomata. They are not tumours of the urachus, but of the fibrous and muscular structures in the space of Retzius. A more interesting, though rarer, variety which occurs in this region must now be considered. These are epithelial tumours, and as in the case of similar types about the bladder apex, five different varieties can be made out: (1) Where adenomatous structures are found on routine examination; (2) Where simple fibro-adenomatous types of larger size have been found clinically; (3) Cases of simple adenocarcinoma; (4) Cases of 'mixed tumours' of unusual structure; (5) Fully developed colloid cancer (adenocarcinoma).

In the whole of the literature only one case each of Groups 3, 4, and 5 has

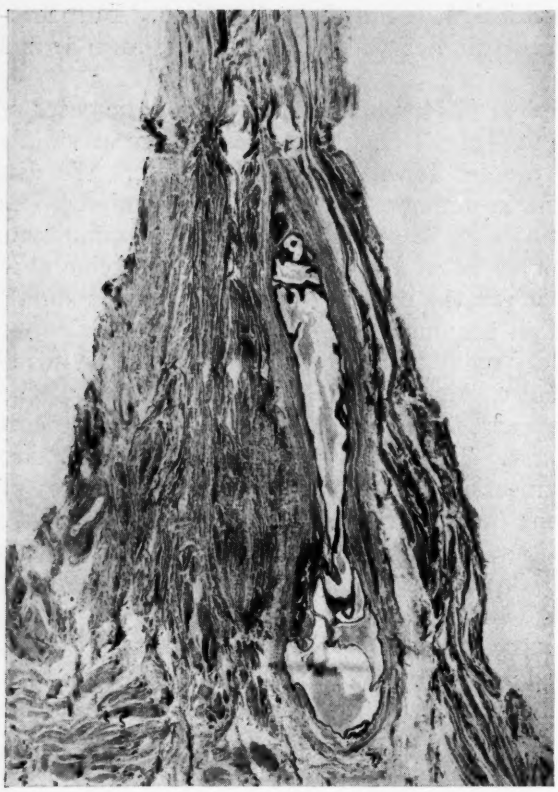

FIG. 349.—Low-power longitudinal section between the umbilicus and the urachus in a child of three (same case as *Fig.* 328), to show an adenomatous structure, probably derived from cells left during the descent of the urachus.

been reported, and all without reference to the etiology here suggested. The conclusion that these growths arose from urachal rests is reached on the following grounds: (*a*) From the nature of their structure, so similar to proved urachal types; (*b*) From their position, in the cases of those found below the umbilicus and above the urachus; (*c*) By a process of exclusion, as they do not conform to growths that could have arisen from any other structure in the region.

1. Adenomatous Structures found on Routine Examination of Specimens.—

A female, 3 years of age, died of tuberculous meningitis. At the post-mortem examination no other lesion except the tuberculous ones were found. The urachus attached to the two umbilical arteries formed an anchor-like arrangement some 3 cm. above the bladder, connected to the umbilicus by a well-marked ligamentum commune (*see Fig.* 328).

An adenomatous structure was found, consisting in the main of two narrow cysts with their long axes from above down (*Fig.* 349). At the upper pole and between the two cysts were glandular-like spaces. A high-power view of the active area in the upper region of the structure is shown in *Fig.* 350. The cyst wall is covered with medium cylindrical epithelial cells whose nuclei are not regularly basal. The acini are also lined by regular low cylindrical epithelium, but in parts there is more than one layer. There are no goblet cells. The cavity has probably been formed by the amalgamation of two cysts. Solid columns of cells and small cysts may be seen at the poles and at various parts of the circumference. In some respects the structure

FIG. 350.—High-power view of part of same section.

resembles an endometrioma. Its general and detailed appearance almost certainly identifies it with similar adenomata as seen at the bladder end of the urachus, while its narrowness and position along the path of urachal descent suggest that it arose from cells shed by the urachal canal. It begins some distance below the umbilicus, and stops short several centimetres above the apex of the urachus. The latter point was verified by serial sections in a downward direction.

The absence of a membrana propria is apparent. This feature distinguishes in general the urachal adenomata from those derived from the omphalo-mesenteric duct.

2. **Fibro-adenoma** (at or near the umbilicus).—Many cases have been reported of adenomata at the umbilicus which correspond closely in structure to urachal tumours. In some cases the tumour was an adenocarcinoma of the rectal type.

In Ehrlich's case there was present at the umbilicus a tumour 3 cm. in diameter. On microscopical examination glandular spaces in a moderately firm stroma were seen. These spaces were lined in parts by cubical, in other parts by cylindrical, epithelium. The author's illustrations show the typical simple fibro-adenomatous structure without basal membrane.

The subject of umbilical growths is too complex to use as a basis for a pathological theory in regard to urachal growths. An extensive literature exists, and may be studied in the writings of Cullen,[28] Kolaczek,[68] Küstner,[71, 72] Ledderhose,[75] Motz,[86] Mintz,[84] Ehrlich,[33] Jores,[61] Ahlfeld,[2] and Pernice.[94]

3. **Adenocarcinoma.**—

Kɔslowski's case[69] (published 1903).—

Male, age 55. A swelling was palpable below the umbilicus. It was described as being in size and shape like the human patella. From it two cords passed—a thick and indurated one from the upper part of the growth to the umbilicus, and a thinner one to the urachus.

Specimen.—In the capsule was a small amount of smooth muscle fibre. In the midst of the somewhat delicate stroma were glandular spaces and acini lined by columnar epithelium of the regular type. The picture on the whole resembled a urachal tumour, and the smooth muscle may have been derived from the obliterated umbilical arteries.

Koslowski considers the case one of tumour of the omphalo-mesenteric duct. Khaum, Cullen, and others considered it as urachal.

I have never seen a membrana propria outside the acini in the proved urachal tumours, and am very doubtful if this can be classified as one. The presence of a basal membrane and the amount of smooth muscle tissue leads me to think that Koslowski was probably right and the tumour was not associated with urachal rests. Unfortunately mere position is no criterion, for, as Schwarz[110] has pointed out, the omphalo-mesenteric duct festoons down below the umbilicus and may be caught up in the abdominal wall inferior to that structure.

4. '**Mixed Tumour**'.—

Adám's case[1] (published in 1929).—

Female, age 43. The patient had a slight elevation of the navel from birth. It started to increase in size shortly before she was examined. The lump was incised, and pieces of tumour commenced to come away. In addition to the primary fungating neoplasm, the growth involved and burst through the abdominal wall half-way

between the navel and the symphysis pubis. The masses at the umbilicus appeared white and translucent. The growths were removed by operation and were found to be anterior to the peritoneum.

SPECIMENS.—The larger tumour, the lower of the two, was 7 cm. in diameter, the smaller one at the umbilicus measured 4 × 3 cm. Both showed on section a stroma infiltrated by carcinomatous cells, and cysts containing mucinous material. Adám thought the lower tumour was primary and the one at the navel secondary. He described them under the name of ' carcinoma planocellulare cysticum '.

It is clear that both tumours arose above the level of the urachus, and from the history it seems probable that the one at the umbilicus was primary. They resemble in structure the cases of Pendl and Barringer, and were probably derived from urachal rests.

5. **Colloid Cancer.**—

HUE and JACQUIN'S case[59] (published in 1863).—

Male, age 45. The patient noticed a smooth tumour in the middle line below the umbilicus. This was incised. Ten months later there was a fungating mass from the navel to the symphysis pubis.

AUTOPSY.—Rectal muscles completely transformed into a gelatinous mass. The parietal peritoneum had undergone a similar change. The tumour involved the bladder apex, and in continuity with it were two colloid masses in the bladder.

This case was probably one of adenocarcinoma originating in urachal rests left near the umbilicus after the descent of the urachus, the growth afterwards spreading by direct descent to the bladder and invading this organ secondarily. It seems evident from the history that the growth started below the umbilicus and spread to the bladder by direct extension. It was probably a colloid cancer, but that it was not a typical tumour is shown by the fact that Laennec considered it a gelatinous cancer, Virchow a gelatinous myxoma, and J. Müller a gelatinous sarcoma (Brady).

From the case-histories given bearing on neoplasms arising from urachal rests, it is evident that we are not treading on such solid ground as when considering the subject of growths of the urachus itself. Clinical material is scanty, but the evidence provided by routine examinations of specimens and by a consideration of the exact structure of the adenomata of the umbilicus gives a good support for the supposition, probable on theoretical grounds, that the urachus during the almost violent nature of its descent and its strong attachment on each side to the umbilical arteries, may spill some of its cells into the transversalis-peritoneal space, and that these are capable of forming tumours of clinical importance.

SUMMARY.

1. On theoretical grounds, colloid adenocarcinomata resembling rectal growths may arise in the muscular wall of the upper part of the bladder.

2. There is definite evidence, both post-mortem and clinical, that such growths do occur as primary tumours, and that the general tendency is for neoplasms in this region to assume the form of colloid cancer.

3. Primitive cells of the urachus are always present in the muscular wall at the apex of the bladder. While these are totipotent, their bias is to form

tumours of the intestinal type. Such neoplasms have, however, certain. distinguishing features of their own.

4. Adenomatous and cystic formations of various kinds are commonly found in connection with the lower end of the urachus, and the connection between these and the colloid cancers can be followed through all the stages.

5. All tumours of the bladder apex must be assumed to be of urachal origin from the point of view of treatment unless the contrary is proved.

6. The treatment is by radical operation, removing the umbilicus and all the tissues between it and the bladder, as well as a large portion of the latter.*

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STUDIES ON HIGH INTESTINAL OBSTRUCTION:

THE ADMINISTRATION OF SALINE AND OTHER SUBSTANCES BY ENTEROSTOMY BELOW THE SITE OF OBSTRUCTION.

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INTRODUCTION.

This investigation was undertaken in an attempt to determine the cause of death in high intestinal obstruction, and to elaborate if possible a means of preventing death after the establishment of the condition. At the outset it is to be emphasized that the study was confined to simple high obstruction, the questions—entirely distinct, as we believe—of strangulation and 'closed loops' not being considered.

Opinion as to the cause of death in high intestinal obstruction is more or less sharply divided. The oldest theory is that death is due to the absorption of some toxic substance formed above the site of the obstruction. The origin of this hypothetical toxin has been the subject of much discussion. There are many who attribute it to bacterial putrefaction of the intestinal contents above the obstruction, where, indeed, both aerobic and anaerobic organisms may be present in large numbers. In particular, attention has been drawn to the possible importance of *B. welchii* in the production of symptoms in intestinal obstruction by the work of Williams.[1] Struck by the clinical resemblance of some of the symptoms of obstruction to those of gas gangrene, he began to investigate, both qualitatively and quantitatively, the occurrence of *B. welchii* in the contents of the obstructed intestine, and to administer antitoxin as a therapeutic measure. As a result of combined experimental work and clinical study he finally suggested that the absorption of *B. welchii* toxin was, in part at least, responsible for the symptoms of obstruction. He also brought forward evidence to show that the proliferation of *B. welchii* and the presence of its toxin could apparently be demonstrated in the human subject and in dogs. The therapeutic use of anti-gas-gangrene serum showed, in Williams's cases, a fall in the mortality-rate from 24·8 per cent to 9·3 per cent. His work excited considerable interest, and serum has been extensively used in the treatment of cases of acute intestinal obstruction.

According to Whipple,[2] however, a toxic proteose is formed in the intestinal mucous membrane itself, part being excreted into the lumen and part passing into the circulation. It is difficult, however, to explain along these lines or along any lines involving the production of a toxin the fact that death follows an obstruction just below the pylorus with great rapidity,

whereas the time of survival is increased when the obstruction is lower down. It would seem, if the toxic theory is correct, that death should be more rapid when there is a greater length of bowel available for the absorption of the toxin than when the absorbing surface is small. Besides those already mentioned, various other sources of the hypothetical toxin have been suggested, including the pancreatic and biliary secretions.

A more recent theory is that death is due to a profound alteration in the chemical composition of the blood—loss of chloride, dehydration, and alkalosis—which reflects upon the physical and chemical processes of the whole body. The chief supporters of this view are Gamble and Ross.[3] Haden and Orr[4] subscribed to the toxin theory, but held that chloride was important in rendering the toxin harmless by a process akin to neutralization. In the course of their experiments they succeeded in keeping obstructed dogs alive for from twenty-one to twenty-eight days by daily subcutaneous injection of 500 c.c. of 0·9 per cent saline. During the progress of the present work White and Fender[5] reported that they had maintained a dog alive and well for twenty-eight days by re-injecting the gastric contents into the bowel below the obstruction. Matsukura[6] succeeded in prolonging the life of a dog with high obstruction to thirty-three days by injection of sodium chloride and water into an enterostomy below the obstruction. Death at the end of these periods might well be attributed to starvation. Among other causes of death in high intestinal obstruction which have been suggested are that it is due to disordered function of duodenal secretions[7] and hepatic insufficiency.[8]

In the experiments to be described in this paper an attempt was made to determine whether, by administration of saline together with energy-producing foodstuffs, life could be sufficiently prolonged to eliminate the possibility of toxæmia as a cause of death. The method of administration—below the site of obstruction—would appear to prevent the possibility of the saline's acting merely as a diluent or neutralizing agent of a toxin produced in the obstructed part of the alimentary tract. Further, on the assumption that the toxin, if toxin there be, is of bacterial origin, experiments were made to find if any correlation existed between the bacterial growth above the obstruction and the condition of the animal.

EXPERIMENTAL METHODS.

Operative Technique.—Healthy dogs of one to three years of age were chosen. On the morning of operation food, but not water, was withheld. Anæsthesia was secured by the open-ether method. The abdomen was opened by a right paramesial incision extending from the costal margin to the umbilical level, and the intestine was brought out into the wound. An obstruction by the 'severed-loop' method was effected at the tail of the pancreas below the lowest pancreatic duct and 10 to 15 cm. from the pylorus. This was the usual site of the obstruction. In a number of cases different positions were chosen, and these are mentioned in the reports on the particular dogs concerned. A transverse incision through the serous coat was made round the duodenum; the muscular coat was divided and reflected by blunt dissection into a proximal and a distal cuff. Two silk purse-string sutures, ½ cm. apart, were then inserted in the submucous coat, and the gut was divided between them. The proximal end was invaginated by further purse-string sutures in the seromuscular coat, and was restored to the abdomen. A Pezzer catheter (size 6),

inserted by Senn's method into the distal opening, was brought out through a stab incision about 3 cm. to the right of the mid-line at the level of the umbilicus. The free end of the distal loop of gut was anchored by sutures to the peritoneum of the anterior abdominal wall, omentum being used to reinforce the apposition. The abdominal wound was closed in layers. The integrity of the obstruction and the absence of infection were, in each case, established post mortem, except in the case of *Dog* 6, which is still living, and in which the condition was inspected at the time of re-establishing the continuity of the gastro-intestinal tract.

Pre-operative Treatment.—The animals were kept in special metabolism cages, and observations were carried out for three or four days prior to the operation. Water was freely allowed, as was food consisting of a mash made of boiled meat, dog-biscuit, and rice. Daily measurement of the body weight was made after the aspiration of the fasting gastric contents in the morning. The fasting contents, withdrawn into sterile containers by means of a vacuum pump, were examined bacteriologically and chemically, as was a second sample of gastric contents similarly withdrawn three-quarters of an hour after a gruel meal (10 c.c. per kilo. body weight). The twenty-four hour urine and stool were collected, and an oxalated specimen of blood was taken.

Post-operative Treatment.—The animal was again kept in the metabolism cage, and was weighed daily. Vomiting, with consequent contamination of urine or stool, was prevented as far as possible by aspiration of the gastric contents two or three times daily, the total amount collected in the twenty-four hours being recorded. Chemical examination of the mixed sample was made daily, and specimens were collected twice a week. The urine passed in twenty-four hours was measured and submitted to chemical analysis. Specimens of stool during the whole post-operative period were mixed and the chloride content was determined. Post mortem the chloride concentration in the skeletal muscles was estimated.

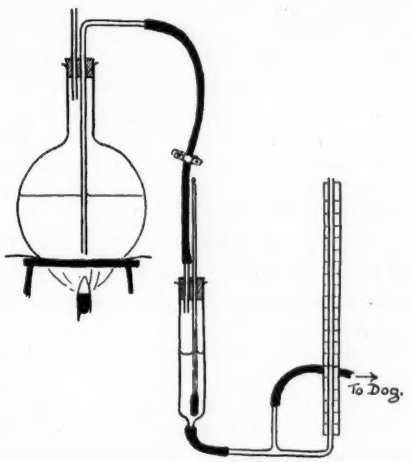

FIG. 351.—Apparatus for continuous feeding by enterostomy.

No food or water was administered by mouth as a rule, but water was sometimes given to cleanse the mouth and throat, or to wash out the stomach, being withdrawn immediately afterwards. Enterostomy feeds were given thrice daily, the first just after aspiration in the morning, the second in the afternoon, and the third at night. In feeding, the apparatus shown in *Fig.* 351 was used. The temperature and rate of flow of the fluid were under control. A manometer connected with the enterostomy tube indicated the pressure in the gut at the site of entry of the fluid into the lumen, thus giving a valuable guide as to the rate at which the fluid should be allowed to enter. Normally, with a manometer tube of bore 0·6 cm., the column varied from 7 to 12 cm. in height above the level of the enterostomy, rising with inspiration and falling with expiration. The optimal rate of flow was approximately a litre in one and a half hours. A few seconds' warning of impending vomiting was given by a steady and rapid rise of water in the manometer, and by immediately stopping the flow of the fluid vomiting could be avoided. Atonicity of the intestine was shown by an exaggerated expiratory excursion of the manometer column; under these conditions it was desirable to reduce the rate of flow. If, however, the mean pressure fell permanently, the rate of flow could conveniently be increased. Mucus in the enterostomy tube had a ball-valve-like action; the column began to rise with inspiration in the ordinary way, but was abruptly arrested, falling suddenly again towards the end of expiration. By slightly increasing the fluid pressure for a few moments the tube could be cleared.

Daily comparison was made between the intake and output of water in order to ensure a sufficiency being given. It was usually necessary to increase the initial daily intake of approximately 1200 c.c. to 2000 or 3000 c.c. at the end of the first fortnight in order to prevent the development of a negative balance in this respect. The daily intake of foodstuffs and the composition of the diet is shown in *Table I.* (The quantities are those in the daily ration, and are distributed among the three feeds.)

Table I.—Composition of Food given to Dogs 4 and 6.

Water	1200–3000 c.c.	Butter	1·00 grm.
Sodium chloride	10–15 grm.	Marmite	0·50 grm.
Glucose	50–100 grm.	Desiccated bile	1·10 grm.
Witte's peptone (trypsin digested)	30–50 grm.	Calcium chloride	0·15 grm.
		Magnesium chloride	0·05 grm.
Cystin	0·2 grm.	Potassium chloride	0·15 grm.
Fresh cream	3 c.c.		

Bacteriological Examination.—With the object of investigating the question of toxin-formation by *B. welchii*, the following methods were employed.

The stomach contents, drawn off with aseptic precautions at bi-weekly intervals, were used for the preparation of a series of decimal dilutions, and equal amounts of these were added to tubes of Robertson's bullock-heart medium. These tubes were examined after twenty-four hours' incubation, and, if the examination was negative, daily for a week. The growth of *B. welchii* in this medium causes a typical viscous haze and evolution of gas. Identification was completed by examination of the morphological characters of the organism present in the cultures and by the production of stormy clot in milk when cultured anaerobically.

In the earlier experiments an attempt was made to estimate the numbers of *B. welchii* in the stomach contents by making shake cultures in Wilson and Blair's medium, but as the medium frequently failed to give satisfactory results the attempt was abandoned. A rough idea of the numbers of bacteria present was obtained by observing the highest dilution from which *B. welchii* were obtained with the use of bullock-heart medium.

Chemical Examination.—In the *blood* determinations were made at intervals of three or four days of the carbon-dioxide combining power, the chloride, and the urea nitrogen or non-protein nitrogen. That the first two of these analyses should be made was obviously essential, since one of the chief hypotheses to be tested was that death in high intestinal obstruction was due to alkalosis and depletion of the blood and tissues of chloride. The blood non-protein nitrogen has frequently been reported in the literature as being raised in cases of high intestinal obstruction, and since the kidney function remains normal, it has been suggested that there is excessive breakdown of tissue proteins in this condition—a conclusion which has also been reached from the finding of a high urea excretion. This suggestion we wished to test.

The *urine* was examined daily. Qualitative tests were made for protein, sugar, and acetone—invariably with negative results—and the daily output of urea and chloride was determined quantitatively. In a few cases the titratable acidity of the urine was determined by titration with sodium hydroxide, using phenolphthalein as indicator.

Before obstruction several analyses were made of the fasting *stomach* contents. After obstruction the stomach contents, collected as described earlier, were examined qualitatively for blood, bile, and lactic acid, and were subjected to quantitative analysis with respect to free hydrochloric acid (the presence of which was confirmed by Gunzberg's reagent), total acid, and total chlorides.

The various methods of analysis employed, all of which are in daily use in this laboratory, were as follows:—

CO_2-combining power	Van Slyke	Blood chloride	Modified Volhard
Urea nitrogen	Urease	Urine chloride	Modified Volhard
Non-protein nitrogen	Micro-Kjeldahl	Gastric chloride	Van Slyke

RESULTS.

Appearance, Body Weight, etc.—The dogs used in the earliest experiments of this series are not strictly comparable with the later ones, owing to the presence of intestinal shunts. In *Dog* 1 the pancreatic and biliary secretions were shunted so as to enter the intestine below the obstruction, whereas in the later treated animals (which, however, received bile in their food), these secretions entered above the obstruction. *Dog* 2, however, received both bile and pancreatic secretion above the obstruction, and behaved exactly as did *Dog* 1, both as regards time of survival and appearance, and as regards chemical phenomena (*Fig.* 352). This result not only shows the validity of treating *Dog* 1 as a control animal in spite of the difference in surgical procedure, but negatives the suggestion previously mentioned, that the biliary and pancreatic secretions are responsible for the production of a toxin in high intestinal obstruction. In the one case any such toxin was poured in above the obstruction and so remained in the intestine for considerable periods, while in the other it appeared below the obstruction and so escaped as in normal animals. Yet the results were identical in the two cases.

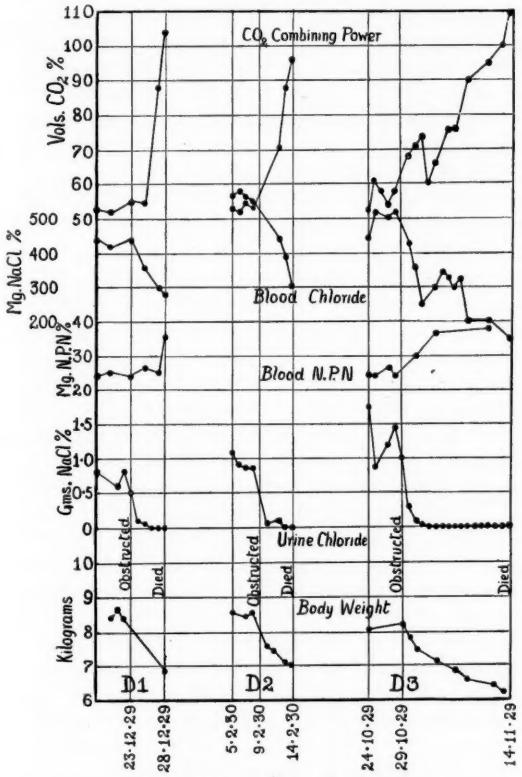

FIG. 352.—*Dogs* 1, 2, and 3.

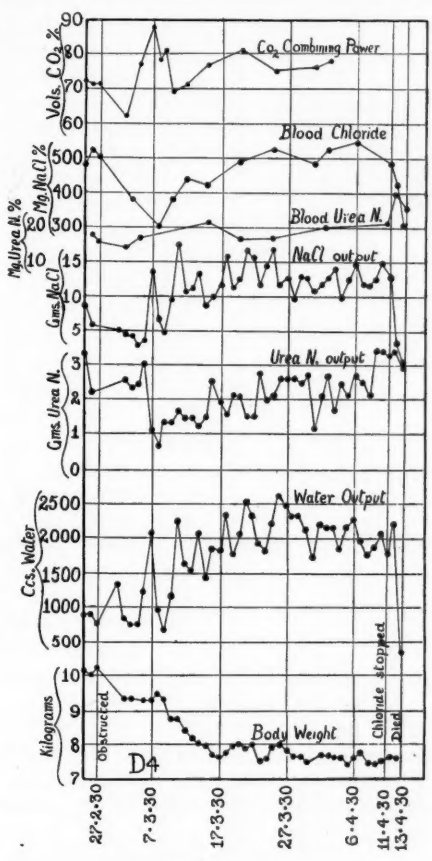

FIG. 353.—*Dog* 4.

Dog 3, like *Dog* 2, received the biliary and pancreatic secretions above the obstruction, which, however, was lower than in the preceding and most of the later animals, being 22 in. below the pylorus. It, like *Dogs* 1 and 2, presented the classical appearances and chemical changes associated with high intestinal obstruction, the differences between it and the other untreated animals being such as are to be expected merely from the different length of intestine above the obstruction—if the chemical theory is correct. *Dog* 3, in fact, was also a good control animal,

for the greater length of gut available for re-absorption of a possible toxin minimized rather than exaggerated the striking nature of the results of chloride administration in the later animals.

Dog 4 is given as a good example of the results of administration of chlorides, water, and foodstuffs. In contrast with *Dogs* 1, 2, and 3, which rapidly became weak and lethargic, *Dog* 4 remained active and mentally alert during the whole six weeks for which treatment was continued. There was also complete absence of the muscular twitching which accompanied the alkalosis of the untreated animals. The body weight, as *Fig* 353 shows, fell somewhat rapidly at first, but shortly after institution of peptone feeding the rapid fall was arrested, and thereafter the weight fell only very slowly. When, however, administration of chloride was stopped (although the other ingredients were continued), *Dog* 4, like its predecessors, soon exhibited the characteristic symptoms of lethargy, muscular twitchings, and weakness, and just before death it suffered the typical ' grand mal ' convulsions.

Dog 5 (*Fig.* 354), which, after surviving twenty-five days, died with an upper respiratory infection, gave very similar results to *Dog* 4, although it was receiving only chloride and glucose in aqueous solution. Even during the last two or three days of life, when the animal was suffering from the extraneous infection which was the immediate cause of death, there were none of the signs associated with approaching death from high intestinal obstruction. There was greater loss of weight than in *Dog* 4, and the progressive loss continued up to death, since, of course, the animal was receiving no nitrogenous foodstuff. Probably, indeed, the general weakness due to semi-starvation was a predisposing factor to the infection. Nevertheless, the results definitely support those of other workers who have found administration of saline to prolong life in cases of high intestinal obstruction.

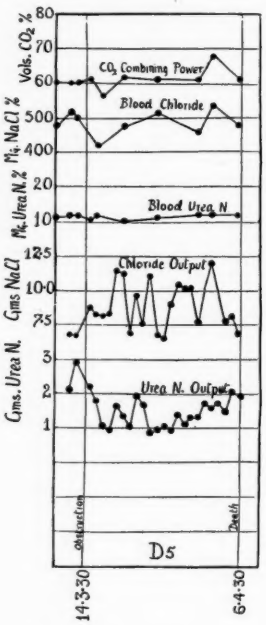

FIG. 354.— *Dog* 5.

Dog. 6 (*Fig.* 355), with a simple obstruction two feet below the pylorus, was treated in the same way as was *Dog* 4. This dog lost weight slowly throughout the period of forty-four days during which the treatment was continued, but he remained bright and active, and showed no symptoms whatever. On the forty-fifth day after obstruction, the intake of chloride was stopped, except for the small amount present in the glucose-peptone solution, administration of which was continued. Two and a half days later he was definitely weaker, less alert, and showed the muscular twitchings indicative of an alkalosis. After chemical confirmation of the state of affairs chloride was restored to the diet, and the symptoms disappeared. Three days later, the blood and urine chemistry being normal, the continuity of the intestinal tract was re-established. At operation it was noted that the proximal intestine was markedly dilated, with muscular hypertrophy, to a diameter of $2\frac{1}{2}$ in.—an important point, as indicating the existence of a stagnant collection of fluid affording almost ideal conditions for the growth of bacteria. Thirty-six hours after the operation the dog was given milk by the mouth for the first time, and during the next four days milk and gruel were given in small, frequent meals. At the end of that time minced meat was given, and as no ill effects were produced, full normal feeding was instituted. The weight remained unaltered until full rations were given, when it began to increase, and at the time of writing the dog is in apparently normal health.

Several other dogs, the details of which are not reported here in order to save space, received the saline and foodstuff treatment by administration below the obstruction. They lived for periods varying from twenty-one to thirty-three days, and in no case could death be attributed directly to the obstruction. A progressive

loss of weight occurred in every case, which treatment was unable to prevent. The dogs which received peptone and carbohydrate in addition to chloride and water undoubtedly lost weight less rapidly than those which received saline alone. Moreover, both *Dog* 4 and *Dog* 6 were in a relatively good state of health forty days after obstruction, a result which is better than any previously reported with chloride administration only. It seems, therefore, that if a really adequate diet (for administration by enterostomy) could be evolved, life could be prolonged indefinitely.

Chemical Analyses.—In those animals in which no treatment was attempted the result of the intestinal obstruction was the production of a most characteristic blood-picture, which, it may be noted, was independent of whether the bile and pancreatic juice entered the intestine above or below the obstruction. With a very high obstruction the CO_2-combining power of the blood began to rise almost at once, though occasionally with a slight delay, due presumably to the acidosis induced by anæsthesia. Simultaneously the blood-chloride began to fall, and these processes continued until, with a severe alkalosis as shown by a CO_2-combining power of 100 vols. or more, and with the blood-chloride at less than half the normal level, death supervened. During the earlier stage of this process the blood non-protein nitrogen or urea nitrogen remained at or near the normal level, and it was only within a day or two of death—some four to six days after the operation—that there was any marked rise in these blood constituents (*Dogs* 1 and 2, *Fig.* 352). An exactly similar picture was obtained in animals with a somewhat lower obstruction, except that the development was slower (*Dog* 3, *Fig.* 352).

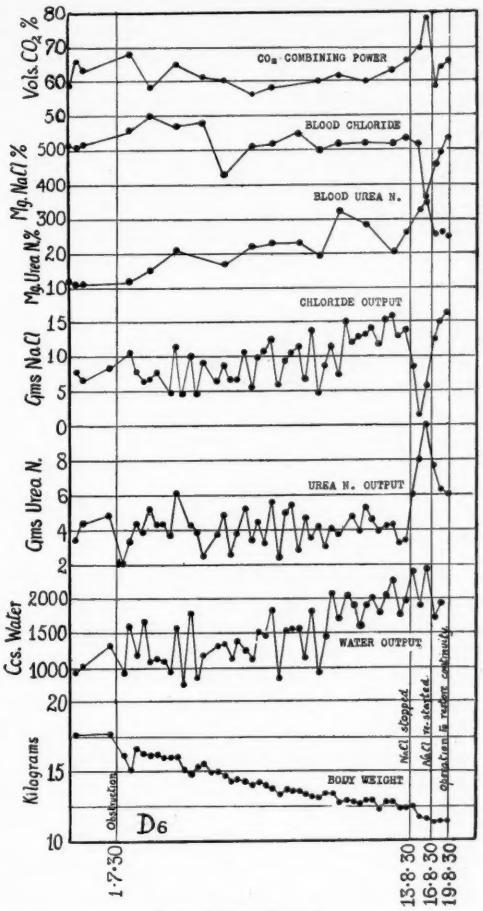

FIG. 355.—*Dog* 6.

The urine of these animals contained, from the establishment of the obstruction, steadily decreasing amounts of chloride, and from about the fourth day after operation it was practically chloride-free. In spite of this absence or low concentration of chloride, the urine, which was somewhat scanty, had a specific gravity rather higher than normal, and urea, in particular, was present in increased concentration. There was not, however, any increase in the total daily output of urea, but on the other hand there was not, as might have been expected when food was being withheld, a very marked decrease.

The gastric contents never showed any significant diminution in chloride concentration in comparison with the pre-operative fasting contents, and although within a few days of the operation free hydrochloric acid had disappeared (or was present only in very small amounts), the total acid was not greatly decreased. It seemed that there was continued secretion of hydrochloric acid (the tests for lactic acid were negative) at little less than normal concentration, but that this acid was almost completely neutralized by mucins, or, in some cases, by the alkaline duodenal contents. The presence or absence of bile, of course, depended on the site of the

obstruction. In those cases in which the bile entered above the obstruction, it was always found in the gastric contents. Lactic acid was never found.

An entirely different picture was presented by those animals which received treatment in the form of saline with or without energy-producing foodstuffs administered below the obstruction. There was no development of alkalosis, and the blood-chloride remained normal. The blood-urea, however, rose slightly in all those animals which were receiving peptone, though not in the one (*Dog* 5) which received chloride alone. Moreover, the increase in the blood-urea became much more marked when the administration of peptone was continued after chloride was withheld (*Dogs* 4 and 6, *Figs.* 353, 355).

As long as the treated dogs received saline, their urine contained choride, the daily output being of the same order as that obtaining before the establishment of the obstruction. When the animals were in equilibrium, the water intake being so adjusted as to balance the output, the daily volume of urine was as great as that of the pre-operation period, and its specific gravity was within the normal range. The concentration of urea was not increased, and in those animals which received no nitrogenous food the daily output of urea was diminished. The animals which were given amino-acids, either as peptone or as pre-digested protein, showed a proportionate but not excessive output of urea. In the stomach contents of the treated animals the chloride concentration remained at the usual high level, the total acid was in normal concentration, and free hydrochloric acid continued to be present, though certainly it was often in lower concentration than during the control period.

When the administration of chloride to one of these animals kept alive by treatment (*Dog* 4) was stopped, death followed in a short time, with the typical fall in the blood chloride, the terminal rise in the urea nitrogen, and the disappearance of chloride from the urine. Similarly, when chloride was withheld from *Dog* 6, the alkali reserve increased, the blood-chloride fell, the blood-urea rose still further, chloride disappeared from the urine, and the usual clinical symptoms associated with death from high intestinal obstruction made their appearance. In this case, however, the feeding of chloride through the fistula was resumed, and coincidently the chemical appearances returned to normal and the clinical symptoms vanished.

Bacteriological Results.—The results of the bacteriological examination of the stomach contents are given in tabular form in *Table II*. They call for little comment. No difference is discernible, so far as occurrence of *B. welchii* is concerned, between the treated and the untreated dogs. It would appear, therefore, that the production of *B. welchii* toxin cannot be regarded as the cause of death in high intestinal obstruction. The fact that occasional specimens in each series show complete absence of *B. welchii* is curious. It seems possible that these specimens were collected from a more acid part of the stomach than the others, and this possible explanation is not negatived by the fact that the same technique was used throughout. Without some such explanation as this the result seems inexplicable.

Table II.—B. Welchii in Aspirated Samples.*

Dog	Pre-operative	Post-operative																	
1	0	0			5	9													
2	No results						Controls												
3	0	0			9														
4	5	3	9	9	9	9	0	9	0	3	0	0							
5	0	0	0	0	0	9	9	9											
6	0	0	0	0	7	0	9	3	0	0	0	9	9	9	9	9	9	9	

*Figures represent greatest decimal dilution in which *B. welchii* was detected. Specimens taken bi-weekly.

DISCUSSION OF RESULTS.

The chemical results obtained from the untreated control animals are entirely susceptible of the classical explanation based on the chemical theory of death in high intestinal obstruction. The secretion of acid into the stomach and its loss by vomiting leads to an alkalosis. The continued increase in the degree of alkalosis even after the stomach contents have ceased to contain free hydrochloric acid is not a serious difficulty, since, as our results show, there is always a considerable amount of so-called 'combined acid' in the vomitus. This combined acid, of course, represents a net loss of chlorine ion to the blood—with the corresponding sodium ion left behind—since it is merely hydrochloric acid combined with some exceedingly weak base, such as protein. The simultaneous loss of mineral chloride in the vomitus naturally reduces the blood-chloride far below the normal level in spite of the attempted conservation of supplies by the reduction or abolition of the urinary excretion of chloride. The loss in the vomitus is not confined to chloride. The water itself is derived from the body stores, and its permanent withdrawal leads to a growing dehydration with, in consequence, a diminished output of urine.

In these untreated dogs our results show no greater output of urea than is to be expected in early starvation—that is, no excessive breakdown of tissue protein which can be regarded as due to the obstruction rather than to mere deprivation of food. The scantiness of the urine, however, leads to a greatly increased concentration of urea in the urine. It is remarkable that the only increase in the blood-urea occurred in those animals which were receiving treatment, and then only in those whose diet included nitrogenous food. It seems fair to conclude that an increase either in the blood-urea or in the breakdown of tissue protein is not an essential feature of high intestinal obstruction. It is true that a blood-urea increase appeared in the two dogs which, after treatment for some time, were placed on a chloride-free diet, but they differed in two important respects from untreated animals. In the first place, their continued loss of weight showed that they were not able to utilize, even if they were receiving, adequate amounts of food from the artificial dietary. Hence they must, at the time of the chloride deprivation, have been nearer the end of their fat and carbohydrate stores than were the untreated animals. Secondly, they, unlike the untreated animals, continued to receive considerable amounts of water and to excrete large volumes of urine. Either of these factors would account for an additional production of urea, and both may have been concerned in the observed increase. However that may be, it seems that some explanation other than the mere existence of the obstruction is required to explain the phenomenon.

The most important question for discussion—at any rate from a theoretical standpoint—is whether the chemical changes in the body fluids are the cause of death in intestinal obstruction, or whether the cause must be sought in some other factor, such as the production of a toxin, the chemical changes being either secondary or of no account. Naturally, the chemical evidence from untreated cases alone is insufficient to answer the question.

It is not known whether a severe reduction of blood-chloride and consequent alkalosis is of itself capable of causing death, though this may be suspected. Even when it is shown that the administration of saline prevents the blood changes attendant upon intestinal obstruction and at the same time prolongs life, one is not justified in concluding finally that death is ordinarily due to these changes. It is highly probable, but it is not completely proved. It still remains possible that the administration of chloride has altered some other factor of which no cognisance has been taken, and that this factor, which develops only when the blood-chloride is lowered, is the real cause of death. Such a supposition seems unlikely, and, in particular, the continued high concentration of chloride in the gastric contents tends to negative the supposition that lack of chloride aids toxin absorption. Still, the possibility remains, though very considerably weakened when one takes into account the observation that in treated and untreated animals alike there is copious bacterial growth in the stomach contents, and equal possibility in both, therefore, of the production of a toxin.

With equal production of toxin, any difference between treated and untreated animals with respect to the production of a toxæmia must lie in the absorption of the toxin. Were toxæmia the cause of death, however, one would expect the reverse of the actual state of affairs; one would expect that the shorter the intestine above the obstruction, the longer would be the time of survival. For the longer the intestine, the greater the rate of absorption, and so the more rapid the development of the toxæmia. Nor does the difference seem to be due to the absence or presence of bile and pancreatic juice, for in our experiments there was no difference (either in time of survival or in chemical and bacteriological findings) between dogs which received these secretions above the obstruction and the dog which received them below.

On the other hand, the fact that the rate of development of the blood and urine picture parallels the time of survival, and is slower with a low obstruction than a high one, is readily explicable on the 'chemical' hypothesis of the cause of death. With the obstruction at the most rapidly fatal site, near the pylorus, the acid secretion of the stomach is lost with little or no chance of reabsorption. Any alkaline secretion there may be, entering the intestine below the obstruction, can be re-absorbed, but if it is re-absorbed it will aid rather than prevent the development of the alkalosis. The lower the obstruction, the greater the length of small intestine available for the re-absorption of the chloride secreted into the stomach, and therefore the less the permanent loss alike of hydrochloric acid and neutral chloride. Hence the low obstruction leads to a slow loss of chloride and a slow development of alkalosis. Since, however, the process is merely slower than with the higher obstruction and is essentially of the same nature, the terminal picture is the same in the two cases.

Though the chemical evidence alone cannot justify the conclusion that death in intestinal obstruction is due to purely chemical causes and not to a toxæmia, the bacteriological and other evidence makes this conclusion extremely probable. The sum total of the evidence does, to our minds, amount almost to absolute proof, and we consider that in the absence of

fresh evidence the hypothesis that death is due to a toxæmia must be held as disproved.

It has been stated by Cooper[9] that " no theory as to the cause of death in high intestinal obstruction has given rise to any practical means of preventing death when the condition has once fully established itself." From the experimental results obtained in this work and applied in the case of *Dog* 6, it seems that success in treatment will depend on the following conditions :—

1. Rapid restoration of the body chlorides and water by subcutaneous and intravenous injection of saline. This should be initiated as early as possible, and may be carried on during operation for relief of the obstruction. At least two litres of physiological saline should be given at this stage.

2. Maintenance of the supply of both chloride and water until re-absorption from the alimentary tract has been resumed, as occurs when continuity is restored by operation. It seems, from the experimental results, that as much as ten litres per day of physiological saline or of Ringer's solution may be required at first, the actual amount being regulated by determinations of the blood-chloride. As a rough guide to the level of the blood-chloride the urine may be tested with silver nitrate and nitric acid ; the appearance of a precipitate of silver chloride indicates that the blood-chloride is near the normal level.

SUMMARY.

1. Death in untreated cases of high intestinal obstruction is preceded by the progressive development of severe alkalosis, by gross lowering of the blood-chloride, and, at the last, by an increase in the blood-urea.

2. For some days before death the urine is chloride-free and the stomach contents contain no free hydrochloric acid, though their total chloride content is normal.

3. It is immaterial whether the bile and pancreatic juice enter the intestine above or below the obstruction. The time of survival is greater the lower the obstruction.

4. Administration of both chloride and water below the obstruction causes the chemistry of the blood, urine, and stomach contents to remain normal, and also greatly prolongs life. Death ensues only after some four weeks and cannot be attributed directly to the obstruction.

5. With peptone and carbohydrate added to the chloride and water, life can be prolonged for seven weeks or more. Withdrawal of chloride then brings about death, with the usual clinical and chemical symptoms.

6. The occurrence of *B. welchii* in the stomach contents is the same in treated and untreated animals.

7. It is concluded that death is due not to toxæmia but to the chemical changes following on loss of chloride and water, and is prevented by their supply.

8. Treatment, therefore, consists in supplying chloride and water until continuity of the alimentary canal has been re-established.

9. There is no evidence that excessive breakdown of tissue protein is an essential accompaniment of high intestinal obstruction.

It is a pleasure to thank Professor D. P. D. Wilkie for the interest he has taken in this work and for valuable criticisms, Dr. W. O. Kermack for suggestions as to the composition of the diet, and Messrs. Duncan & Flockhart for the free supply of proteolytic fluid and desiccated ox-bile. One of us (T. C. M.) holds a Grocers' Company Scholarship, and two of us (C. P. S. and D. M. D.) are in receipt of part-time grants from the Medical Research Council. In addition, one of us (C. P. S.) holds a Carnegie Teaching Fellowship. The expenses have been partly defrayed by a grant from the Moray Research Fund of Edinburgh University.

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VICARY'S PREDECESSORS.

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(Being the Vicary Lecture delivered at the Royal College of Surgeons of England, on November 6, 1930.)

Thomas Vicary, whose name we honour to-day, was Serjeant Surgeon to King Henry the Eighth. This lecture was founded by the Barbers Company to commemorate a man who was Master of their Company and who held a distinguished position in Surgery. His portrait hangs on the wall of this College, his life has been described in happy manner by his biographers, and, though he lived four hundred years ago, our feelings towards him are of intimacy and friendship. Of his predecessors in the office of King's Surgeon, much less is known, and though they must have been men famed in their day, the majority have faded into obscurity. The object of this paper is to rescue them from oblivion, and to put on record the names, at least, of these men who were chosen to attend their sovereigns in peace and in war, and who by their work contributed to make British Surgery what it is.

Unfortunately their biographies cannot be given in great detail, for the lives of medical men in mediæval times have to be drawn up from varied and all-too-scanty sources. There is no book in which the names and services of these men are gathered, but only indirect references scattered through the dusty files.

The source of much of the information gathered for this lecture has been taken from the various Rolls. The first important series of these are the Pipe Rolls, on which were entered particulars of sums of money paid out by the Exchequer. Then there are the very important records known as the Patent and Close Rolls, which form a continuous series from the reign of King John. On these were enrolled copies of the letters-patent, that is, those open and public, and close letters, meaning private letters, which were sent out in the King's name on all sorts of business. These may be described as the equivalent of the duplicate correspondence files of modern government departments. These letters were written on parchment skins, which were sewn together end-to-end and then rolled up. Hence is derived their name of 'Rolls'. These Rolls have been transcribed and printed and so made available for students. They deal with an infinite variety of business, ranging from grave affairs of state to petty details of rewards and punishments to faithful servants or erring subjects.

Among this mass of material one finds the scanty notices of our early predecessors. These notices, generally brief, commonly refer to some grant of land or money conferred on them as wages or rewards for services rendered, and it is for this reason that their names have been preserved.

From these and other similar sources a continuous sequence of medical officers of the reigning sovereigns can be traced back as far as the time of Edward the Confessor. Sometimes merely the names are recorded, but here and there a few details emerge, trifling details for the most part, but enough to throw a little light on the distant past.

In the eleventh century, in which our first record comes, medicine was largely, if not entirely, the prerogative of the Church. Learning was confined to the monasteries, and the aspiring cleric learnt medicine along with grammar and philosophy as one of the humanities. Thus, Abbot John of Cella, of St. Albans, was eulogized as " a Priscian in Grammar, an Ovid in Verse, and a Galen in Physic ". Nor did these early physicians confine themselves to the practice of medicine. They were first and foremost clerics, and for their medical services to their sovereigns they were commonly paid by gifts of livings.

The first of Vicary's predecessors to be recorded here is BALDWIN, the Abbot of Bury St. Edmunds, who was physician to Edward the Confessor. He was a cultured man, and described as " gretly expert in craft of Medycyn ".

Following him came NIGEL, whose name figures in Domesday Book as the recipient of various gifts of land. He was physician to William the Conqueror. There is no record of his presence at the Battle of Hastings. The Bayeux Tapestry gives us some idea of the treatment of the fallen (*Figs.* 356–359).

William Rufus had as his physician JOHN DE VILLULA, a learned divine, coming, it is believed, from Tours. He became Bishop of Bath, where he founded a monastery which became a seat of learning.

During the reign of Henry the First, the Pipe Roll mentions the names of GILBERT DE FALAISE as 'Medicus'; of GRIMBOLD who lived in the Royal Court; and of CLARUMBOLD who was physician and chaplain.

In the reign of Stephen appear the two shadowy names of ERNOLF and IWUD. Little is known of these beyond the fact that their names appear as witnesses to certain documents.

The Pipe Roll of the reign of Henry the Second includes the names of a number of medical men, such as JOHANNES medicus and RUDULFUS medicus, and there is the faint echo of one tragedy in a terse record that MASTER RALPH DE BELLOMONT, King's physician, was drowned in the Channel in 1170.

Norman Moore described MAGISTER RAMULPHUS BESACE as physician to Richard the First in Palestine, and George Parker, in his History of Surgery, quotes a reference from the Pipe Roll to the effect that MASTER JOHN OF BRIDPORT, medicus regis, should have a suit of clothes when he went to Normandy.

The reign of King John is not productive of much information, though there is one note in the Close Rolls ordering that the necessaries for three horses should be provided for MASTER R. FUKET,[1] the King's physician, which shows that even in these early days some arrangements were made for the provision of clothes and necessaries for the physicians attached to the King, but it is not till the reign of Edward the Second that we find an orderly account of their rights and duties set out in the Household Ordinances.

It will be noticed that up till now there has been no mention of surgeons,

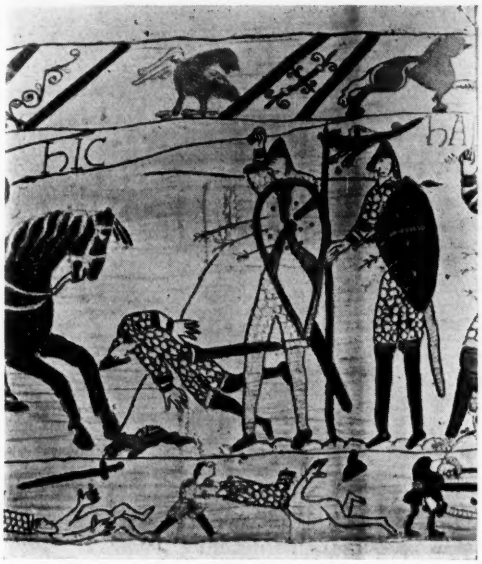

FIG. 356.—The Battle of Hastings from the Bayeux Tapestry. The scene depicts the day going against the Saxons. The lower border shows how the fallen were stripped of their armour.

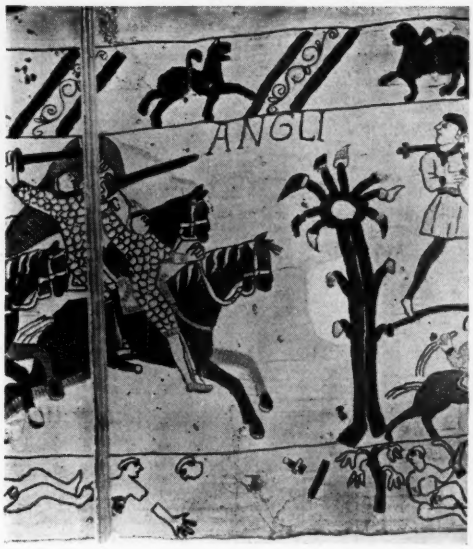

FIG. 357.—The last scene from the Bayeux Tapestry. The Saxons fly pursued by the victorious Norman horsemen.

FIG. 358.—Enlargement of the lower border of *Fig.* 356. The liveliness of the picture indicates that the men were stripped while still alive.

FIG. 359.—A figure from the lower border of *Fig.* 357. This shows a wounded man, stripped of his armour, sitting up and holding a piece of mandragon, the well-known narcotic herb used in the Middle Ages.

but only of physicians. It has to be remembered that in these early days doctors were physicians and surgeons too, for at that time medicine had not thrown away her right hand of surgery. The reasons why surgery was divorced from medicine—a fact we still deplore—and the date at which the separation took place, are still obscure, though the subject has been hotly disputed. Suffice it here to say that the separation did take place, and probably sometime during the twelfth century.

It is for this reason that we do not find records of a separate appointment as a surgeon to the early kings. In fact it is not till 1233, in the reign of Henry the Third, that the scanty records yield a notice of a medical man having the title of Surgeon to the King. The first of these surgeons, as far as I have been able to discover, was Master William.

MASTER WILLIAM.—In the Calendar of Patent Rolls given at Woodstock on 12th May, 1233, in the 17th year of the reign of Henry the Third, appears the first notice of an appointment of a surgeon to a king.[2]

> **Presentation to Master William, the King's Surgeon to the Church of Houkerinton, in the same diocese (Lincoln) in the King's gift by reason of the land of Thomas, son of Walter, being in his hands.**

From this scanty notice it can be inferred that William was an educated man and of standing in his profession, seeing that he held the title of Master; also that he was a cleric, and that he was being rewarded for his services by presentation to a living. More than that one cannot say, for with this solitary reference he appears and disappears in the pages of history.

Almost equally obscure is the second surgeon of whom there is record, namely, MASTER DAVID. In 1241 there was a mandate from King Henry the Third to the Justiciary of Ireland to provide, as soon as opportunity offer, Master David, the surgeon, with 100 shillings of land in escheats or wards.[3] Again, two years later, in 1243, an order was given to the Justiciary of Ireland to give possession (*seisinam*) to Master David, the surgeon, of certain property of John of Kaerdiff.[4] Master David must have died about this time, and in not too flourishing conditions, because a few years later, in 1248, King Henry made an order, bestowing on Alice, the widow of Master David, formerly the King's surgeon, for her maintenance during her life, those three obols (*obolos*) which Brother Nicholas, the Westminster recluse, used to have through the charity of the King.[5]

FIG. 360.—Henry III sailing home from Gascony, 1243. Drawn by Matthew Paris. (*From Green's "Short History of the English People", by kind permission of Messrs. Macmillan & Co. Ltd.*)

After the death of Master David it would appear that his successor was MASTER HENRY DE SAXEBY. At any rate we hear of him first in 1251, and he is notable among other things for being the first surgeon to carry the title

of Serjeant Surgeon. It is difficult to be sure of the exact meaning of the title in these times. The first reference to it is as follows :—

> **Grant to the King's serjeant, Master Henry, the King's surgeon, King's clerk of £10 at the Exchequer etc.**[6]

The reading of serjeant in the Latin in which the Rolls were written is 'serviens', that is to say 'servant'. The King had many such serjeants attached to his person ; thus there was a serjeant of the napery, a serjeant-at-arms, and a serjeant of the pavilion. It seems probable that at this time the description meant that such a one was attached to the King's person and received grants of land or livings in lieu of wages. Later it appears to have meant that, when more than one surgeon was attached to the monarch, the senior of them was termed 'Serjeant Surgeon' and was responsible to the King for the surgery of the Court and for providing and keeping the necessary drugs. Be this as it may, Master Henry de Saxeby seems to be the first of the long line of Serjeant Surgeons which came down through Thomas Vicary and is continued to this day in the person of Sir Hugh Rigby, K.C.V.O.

Now for the first time we hear what was the salary of the King's surgeon. This was laid down in the case of Master Henry and was £10 per annum, which he was to receive at the Exchequer until the King could provide for him in land to that yearly value to hold for him and his heirs.[7] This promise apparently was made good, for about the same time appears a notice that Master Henry had served the King well, and the King, desiring his promotion, commanded the Justiciary of Ireland to provide 10 librates of land.[8]

There is one little incident of Master Henry de Saxeby's life that has been preserved, and is of interest as showing the manner in which working men were treated in those days. In 1251 a wall in Westminster Hall collapsed, and in its fall injured seven men. Master Henry was detailed by the King to look after these men, and the King paid also for their food, attendance, and necessaries for their cure.[9]

The most remarkable surgeon of the period was Master Thomas de Weseham, who must have been a man of considerable attainments and character, for he held the position of surgeon to the King for some twenty years. He has been well known for many years to coin collectors, but little has been said of him as Serjeant Surgeon to Henry the Third.

Of his birth and education unfortunately little is known, though it may be inferred that he was a layman and not a cleric, and that from his name he was probably an Englishman. He may have taken his name from the little town of Weseham in Lincolnshire, and he was at any rate a contemporary of the famous Roger of Weseham (d. 1257), who was Lecturer on Divinity at Oxford, and later Bishop of Lichfield. It is pleasant to think of the possibility of these two men being brothers, one an eminent divine, and the other a distinguished surgeon.

We first hear of Master Thomas of Weseham in 1253, when he is cited as amongst those accompanying the King in one of his expeditions to Gascony.[10] It seems probable that he went in the capacity of body surgeon to the King, and that he had as his companion Master Patrick de Carliolo, the physician. The accompanying picture (*Fig.* 360) is Matthew Paris's idea of how they came home.

After the return from France several gifts of land were bestowed on Master Thomas. In 1255 he was given a house in Colchester which had belonged to Isaac, the Jew,[11] and the following year was granted for life the bailiwick of the Forest of Cannock.[12] In succeeding years he received other grants, but one is of special interest and unusual. In 1260 the King granted Master Thomas a die in London, that is, he was authorized to mint coins, and presumably there was a certain amount of profit attached to the right.[13]

FIG. 361.—Aaron of Colchester. Forest Roll. Public Record Office. (*From Green's "Short History of the English People", by kind permission of Messrs. Macmillan & Co. Ltd.*)

Mr. L. A. Lawrence, a Fellow of this College, possesses a coin minted by Weseham, and by his kindness it is reproduced here (*Fig.* 362). The reading on the coin is: 'Henricus Rex III' on the obverse, and 'Thomas on Lund' on the reverse side.

For twenty years after the gift of the die, Master Thomas of Weseham and his wife, Sarah, lived in the Parish of St. Michael in Bassieshaw.[14] It is pleasant to think of this honoured surgeon spending his declining years in peace and quiet in the City of London.

It would seem that Weseham was succeeded in the late years of Henry the Third's life by another surgeon, namely, MASTER SIMON DE PRESTON. There is little to record about him beyond the fact that in 1271 he was exempted for life from being put on assizes or from being made sheriff against his will.[15] Thus early in mediæval times medical men were granted privileges similar to those which are enjoyed by members of this College to this very day.

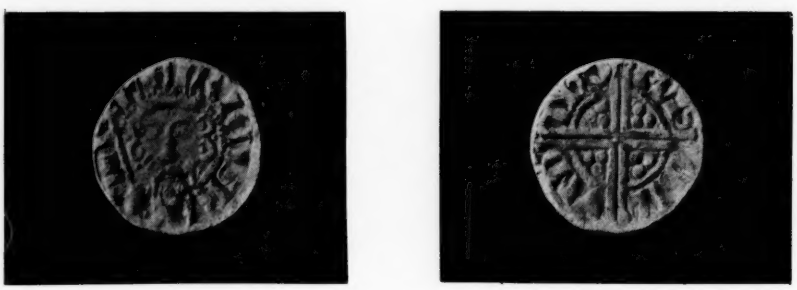

FIG. 362.—A coin minted by Thomas de Weseham, Surgeon to Henry III. (*In the possession of L. A. Lawrence, Esq., F.R.C.S.*)

The long reign of Henry the Third is thus notable for the appearance of surgeons, as apart from physicians, and also for a great spurt in the study of medicine. All over the continent of Europe hospitals were springing up, and the same was happening in England. Henry was a Frenchman, and his Queen, Eleanor of Provence, was a Frenchwoman. People complained that he filled the chief posts of this island with a horde of hungry relatives. Certainly many French doctors came over, and no doubt brought with them learning from the French Universities which was of inestimable value to us.

Not only did Henry the Third have his own medical and surgical attendants, but he provided a medical establishment for his Queen, Eleanor of Provence, and also for his son, Edward, later Edward the First, and probably most of these were Frenchmen.

Edward the First was cast in a different mould from his father, Henry, and during his long minority his character was so formed that by the time he became King he had acquired a reputation as a statesman, organizer, and general second to none in the kingdom.

It is during this reign that we get the first glimpse of an ordered household, with list of the duties and emoluments appending to the various offices, and, according to Withington, the germ of an organized army medical service. Indeed, Garrison gives Edward the credit of making the first attempt at the formation of a military medical service in the Middle Ages, apart from the Byzantine Empire.

Edward the First had two notable surgeons, MASTER SIMON DE BEAUVEYS (de Belvaco), and his son, PHILIP. One may suspect that Master Simon was a Frenchman, taking his name from the town of Beauvais in France, but there is nothing to tell about his early life or where he got his training. His son, Philip, was probably brought up in England and spent his early life about the Court, entering the King's service at an early age, for in 1279 he is described as King's yeoman and receives a grant of 200 marks to get married.[16]

Many grants of land were made to these two, which it would be wearisome to relate. The share, however, which Master Philip took in Edward the First's campaigns against Scotland is of interest as illustrating the manner in which war was then waged.

Of the five campaigns against Scotland, those of 1298 and 1300 are of special interest, because in the records of these are the earliest indications of anything like an organized army medical service.

In order to make the matter clear it is necessary to give a short account of these expeditions.

In 1298 Edward the First led an expedition against the Scotch who were ravaging the North of England. The army consisted of cavalry and infantry, led in its several companies by bannerets, and was divided into four battalions, the third of which was commanded by the King himself. Besides bannerets the host included simple knights and valets, who appear to have been in rank equal to esquires. It was the practice to appraise or value the horses of knights and others at the time when they joined the army, and to pay for such of them as were killed or lost in the King's service; and it is from these lists that some of our information about the doctors is derived.

The English forces were not opposed until they came to Falkirk, where they found Wallace and the Scots drawn up in battle array behind a morass, and the Battle of Falkirk was fought on July 22, 1298. It was summer time, and the night before the battle the English troops lay on their arms, and the King laid his long shanks on the bare ground beside them. During the night a sudden alarm startled the horses, and the King's charger trampled on him and broke two of his ribs. This accident, however, did not prevent the King leading his troops to battle and victory. The Scots were defeated with very

great loss, while the English lost only a few men. After the battle was over, King Edward, feeling no doubt in pain from his injury, repaired, according to Rishanger, to Stirling, and lodged in the Convent of the Black Friars to convalesce.[18] According to Comrie, however, King Edward went for his convalescence, not to Stirling, but to the Priory or Hospital of Torphichen, situated a few miles away, and which still stands to this day.[19]

It seems more likely that he should have gone here, because the Priory belonged to the Knights Hospitallers of the Order of St. John of Jerusalem, probably an off-shoot of the well-known Priory in Clerkenwell; and moreover the head of the Hospital was an Englishman, Alexander de Welles, who was killed at Falkirk fighting on the English side.

There were a certain number of medical men included in the lists of those present with the King. First there was a physician, MASTER JOHN DE KENLEYE, who is mentioned as having letters of protection on account of being about to set out to Scotland in obedience to the command of the King (*in obsequiem regis*). In the Roll of the horses of the men attached to the royal household, Master John de Kenleye is also noted as having a horse, dappled or iron-spotted, valued at 12 pounds; also that he had a valet (*vallettus*) who had a horse, iron-grey, valued at 6 marks. The valet's name was William le Mareschal.

It is not clear as to the meaning of the word *vallettus*. It may mean that the physician was accompanied by one of his own profession, or it may mean that he was a soldier detailed to protect and serve his master.

The only surgeon who is described as being present is MASTER PETER (*Cirurgicus Regis*). He had a horse with a star on his forehead valued at 16 marks, and his valet, John le Mareschal, also had a horse valued at 16 marks.

It is uncertain whether Philip de Beauveys was present in this campaign: probably he was, and possibly it was he who diagnosed the fracture of the King's ribs.

As far as can be ascertained no arrangements were made for the care of the sick and wounded, and there are no details about wounded men.

The Scotch Campaign of 1300.—In April, 1300, Edward conducted a successful campaign against the Scotch. Fortunately for us the Wardrobe account of this, the 28th year of the reign of Edward the First, has been preserved, giving detailed accounts of disbursements. This includes many interesting details concerning the medical men who were present, their pay, the clothing they received, and the horses they had.[20]

The actual facts, as far as they can be ascertained, are that the King had in his train, his physician, Master John de Kenleye, with two servants, or perhaps juniors (*valetti*); also his surgeon, Master Philip de Beauveys, and two assistants (*socii*); and in addition there was Master Peter, who was noted as being present in the campaign of 1298. The King's physician and surgeon each received a knight's pay, two shillings a day, and the others who ranked as esquires half that sum. In addition to their pay, the King's physician and surgeon each received four marks for clothes for summer wear and four marks for clothes for winter wear, and while they were with the Court they received their board and lodging free.

Master Peter, who seems to have been a junior officer, only received one mark for clothes instead of four, as his seniors did.

It is open to argument whether the four men described as '*valetti*' and '*socii*' were really medical men, as has been supposed by Withington. They may have been, but there is nothing to substantiate the theory.

FIG. 363.—The chatelaine administering mandragon to a wounded knight. (*Published in "Chirurgiens et Blessés", by Cabanes.*)

In support, however, of the theory of an organized medical service, it should be stated that Master Philip, the surgeon, was allowed the sum of 40 shillings annually for the purpose of supplying diverse medical stores for the use of members of the household who might be injured, and Master Peter was allowed half that sum. Master Philip must have been hard worked during this campaign or have had bad luck, for he lost three horses in the service of the King, for which loss he was compensated at the rate of 40 shillings a horse. The records do not say whether these horses were killed in action or died of disease.

FIG. 364.—A wounded knight transported by a comrade to a monastery. (*Published in "Chirurgiens et Blessés", by Cabanes.*)

It is not quite clear whether Master Kenleye, the King's physician, was present during the whole of the campaign or not. For part of the time he was sent off by the King to Caversham to attend the King's daughter, the Countess of Gloucester, who was ill. During the time he was away from the Court, Master Kenleye received one shilling a day.

A careful search has been made to discover what arrangements were made for the care of the sick and wounded in these campaigns, but practically nothing has come to light. There is evidence that some sort of consideration was given disabled soldiers, for it is recorded that a certain Harvey de Cornubia, a valet of the King's napery, who was wounded by the Scots in Galway, was given his expenses to pay for his return to England and for his necessary medicines.

But as for the immediate treatment of the wounded, nothing seems to be known. *Figs.* **363, 364,** taken from the French romance of Lancelot du

Lac, would seem to illustrate the sort of thing that happened. Here is a wounded knight tended by a fair lady, an early prototype of a nursing sister, who by her association with mandragon obviously was accredited with a knowledge of drugs and salves. The second picture shows a wounded knight taken by a comrade to a monastery to be cared for by the hospitable monks.

Some such methods as these we may suppose were used, for there is no hint of organization for the care of the wounded. We have to wait for nearly two hundred years, till the time of Queen Isabella of Spain, for the establishment of Camp Hospitals and an Ambulance system.

Edward the First died in 1307, while leading yet another expedition against Scotland, but his death only arrested for a moment the advance of his army. Edward the Second, however, had neither his father's skill as a general nor his gift to govern. As is well known, the Battle of Bannockburn, in 1314, resulted in the total defeat of the English, the King narrowly escaping with his life. Very few details of this disastrous battle have been preserved, and we know nothing of the medical men who may have been present or of any of the medical arrangements. After this campaign a number of inconclusive expeditions resulted, in 1323, in a thirteen years' truce between the two countries.

One of these expeditions, however, that of 1322, deserves a special mention, not on account of its military importance, but because, among the few details which have been preserved, there is a record of the medical stores which were sent up from London to Newcastle by water for the use of the troops by MASTER STEPHEN of Paris, the King's surgeon.

This document, the earliest of its kind, proves beyond doubt that there was at this time some sort of organization to provide for the medical needs of the army, and that some person or body of men were entrusted with the preparation and despatch of the material. In this instance it looks as if the person whose duty it was, was the King's surgeon, Master Stephen of Paris. An examination of the document shows that the drugs were packed in two great 'paniers', which cost 6d. apiece, and were despatched from London to Newcastle by ship, and thence to Edinburgh by water, and from there back again to Hayleland. The total cost of the goods, including carriage from London, was £91 3s. 9d. It is interesting to note that the term 'panier' still survives in our army, and that it is in them that the medical stores of the Field Ambulances are carried to this day.[21]

The stores themselves, called " drogueries and implastra ", are specified minutely, and include 3 lb. of oxerocrosin; 3 lb. of dyataroscos; 6 lb. of apostolicon; 2 lb. of Saunguys Draconis, and various other forms of ointment.

Of further details of the King's surgeon, Master Stephen of Paris, we have none. Beyond the bare mention that he was the surgeon, and that MASTER ISAMBERT was the physician, the records are silent.

Though so little is known about the medical men of this reign, we have some very important details about the offices and emoluments of the King's physician and surgeon. These are included in the Household Ordinances of Edward the Second, and there we find them set out clearly and definitely for the first time.[22] Whether they were so set out and defined by Edward

the Second is not certain. One suspects that they date back to earlier reigns, though there is no direct evidence as to when they were first drawn up.

The Ordinances were written in old French, and the following is a modernized version :—

> *A Cyrurgien.*—The surgeon shall have his diet every day in the hall, if he is not hindered by some business certified before the Steward and Treasurer. And then he shall have his livery as a knight of the household, whether he be well or ill, that is to say, two darres of bread, one pitcher of wine, two messes de gros from the kitchen, and one mess of roast. And shall take every day for his chamber, one pitcher of wine, three candles, one tontis, litter all the year, and fuel for dinner time, of the usher of the hall. He shall have 12 pence a day wages until he be advanced by the King and two robes yearly in cloth, or 8 marks in money. For things medicinal he shall have forty shillings by the year.

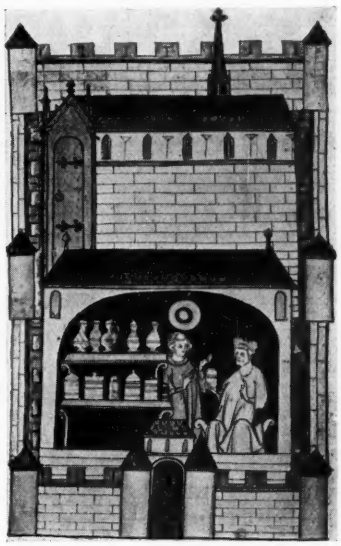

FIG. 365.—An apothecary's shop. Thirteenth century. (*From the "Treatise of Surgery", by Roger of Parma, Sloane,* 1977.)

There was a charge for the physician similar in every way, except that the physician was allowed three darres of bread instead of two, and he was allowed wages for three servants.

It will be noticed that the surgeon was allowed forty shillings for the provision of medical stores, and that this was the same sum as was allowed Master Philip de Beauveys for the same purpose in the reign of Edward the First. It looks as if it was the duty of the King's surgeon to provide medical stores for the use of the members of the royal household. The accompanying pictures are illustrative of approximately contemporary customs (***Figs.* 365–367**).

FIG. 366.—The dentist. Fourteenth century. (*Roy. MS.* 6 *E.VI.*)

FIG. 367.—Bleeding. (*From the "Luttrell Psalter" f.* 61.)

The reign of Edward the Third rings with stories of tossing spears, of gleaming armour, and blazoned shields, stories Froissart loved to chronicle. A glamour still clings round the name of Crécy, though nigh six hundred years have passed.

The battlefield of Crécy remains to this very day very much as it was in 1347. It is easy to mark the position taken up by the small British Expeditionary Force—" that contemptible little army "—and the slope of ground where they withstood the chivalry of France, and the very site of the windmill under which the King stood watching the Black Prince win his spurs.

Who were the doctors at this battle, and what position did they occupy during the fight? Were they with the little group which stood round the King under the old windmill, or were they with the carts which we know were parked not far away in the rear? Their number and their position must be matter for conjecture only, because the records are lamentably lacking in all such details.

It is known that the King's surgeon at that time was MASTER ROGER DE HEYTON, and though it is not definitely stated that he was present at the battle, it is probable that he was; and it is most likely that he had at his command medical stores packed in the service paniers which have been mentioned as being provided in the reign of Edward the Second.

At the time of the Battle of Crécy, Master Roger must have been a man of mature years, for he had then been some twenty years in the King's service. The first reference to him occurs in 1328, in the second year of the reign of Edward the Third.[23] He was then described as the King's surgeon, and was granted at the request of Edmund, Earl of Kent, custody of the town of Pennanthlu in Merionethshire. In succeeding years he received a number of grants of land and otherwise, and in one of these grants it is specifically stated that it was in consideration of his services in staying constantly by the King's side. We know also that he received as a gift a house close to the gate of the Palace of Westminster, where he lived with his wife, Isabel, and his two daughters, Alice and Isobel, who were aged 19 and 18 respectively, when he died on May 13, 1349, two years after the Battle of Crécy. The exact date of his death has been preserved on account of a dispute which arose later as to the ownership of this house.[24]

Whether Master Roger took part in the Battle of Crécy must remain in doubt. It seems clear that he intended to partake in the expedition, for in the French Roll, dated May 15, 1346,[25] is preserved a writ to the Bailiff of St. Albans: " to exonerate the King's beloved Magister, Roger de Heyton, the King's surgeon, who was about to set out on the King's service, from any contribution to find armed men on account of his land and tenements in that town."

One cannot refrain from making mention here of the physician who was contemporary with Master Roger, namely, MASTER JORDAN OF CANTERBURY, the King's physician, who certainly was present at Crécy. It is recorded of him that he received as wages of war 109 shillings for a period of three years and a half and 56 days. Master Jordan died about 1361, after some thirty-five years' service with the King.[26]

Search the records as one will, no mention is made as to the method of picking up or treating the wounded, except a brief reference by Joshua Barnes,[27] in which he quotes Giovanni Villani in his description of the events following the battle :—

> and so the High Mass of the Holy Spirit was solemnly sung and the Victory acknowledged to the Author thereof. Moreover for the sake of the Dead the Pious King caused the place of the Battle to be consecrated for the decent sepulture of his enemies and friends: and that the wounded being removed from among the dead, should be dressed; to the meaner sort of whom he afterward gave money and sent away.

Who dressed the wounded, and how they were taken away, we do not know. It is stated that there were a certain number of surgeons attached to the Welsh troops, and they may have done the work. As regards transport, we know also that Edward had a certain number of carts which were parked at the rear during the battle, and we may suppose that any wounded that were carried away were taken in these carts. This was the universal way of transporting the wounded, even in the time of the wars of Marlborough and up to the Napoleonic era.

That there was the germ of a mechanism for invaliding soldiers no longer fit for active service, and some sort of pension scheme, is shown by the following record. A writ of protection was given to the Sheriff of the County of Northampton, in favour of Sir Nicholas de Burnaby, who had served in the retinue of Edward, Prince of Wales, and had been severely wounded in the foot by a quarrel. There is also a similar writ in favour of Sir Peter Malore, who had appeared personally in chancery and who was manifestly so maimed that he could no longer give aid in warlike actions; and another writ to the Mayor of Cambridge, stating that William Brice had returned to England grievously injured, and by an inspection of his wound in chancery it was evident that he could no longer serve usefully.

Though our government records do not throw any light on the method of treating the wounded, there is an illuminating passage in Froissart which is probably a correct picture of the times.

It concerns the famous knight, Sir John Audley, who fought valiantly at the Battle of Poitiers. He was wounded in many places, and when overcome by them, he was led or carried out of the press by his four squires who laid him under a hedge, disarmed him as gently as they could, in order to examine his wounds, dress them, and sew up the most dangerous.

Who taught these squires how to bandage and treat wounds? I suspect that the chatelaines—the wives of the knight—did, and that part of the duty of the squire was to carry dressings and salves.

The last of the surgeons to King Edward the Third was Master Adam Rous, who was first mentioned in this capacity in 1359. He was granted a property in the Poultry, where he seems to have lived and died in 1370. His will has been preserved.[29] From it we learn that he had a wife, named Juliana, and a brother, William. To Sir William Stodeley he left a girdle with pouch and knife, given him by the Duke of Lancaster, and among other bequests he gave some house property to the Priory of St. Bartholomew. From this I would like to assume that he was a good Bart.'s man,

having received his training in the old Priory, but as regards this there is no information.

It would not be right to leave the reign of Edward the Third without a mention of the great English surgeon, JOHN OF ARDERNE, who has given us our first book on Surgery, and who no doubt knew the men we have been describing.

Richard the Second had three or four surgeons. It is pleasing to be able to record a kindly action of this rather poor King towards one of them, namely, JOHN LECHE. We hear of this man first in 1382, when he is described as King's surgeon. Then in the following year appears this record :—

> Grant, for life, to the King's surgeon, John Leche, whose sight is so weak that he can no longer labour in his office, of the wages and robes yearly as an esquire in the king's household, whether he is present or absent therefrom, as heretofore received by him from the keeper of the wardrobe.

As late as 1391 he is still described as the King's surgeon, and is given a grant of land and is to continue to have his robes or 40 shillings in lieu thereof.[30]

At least two other surgeons served Richard the Second, namely JOHN SALESBURY and WILLIAM BRADWARDYN. Of the former, John Salesbury, there is just a bare record in which he is described as surgeon, the King's serjeant (*serviens noster*), and that he was granted 40 shillings for life at the exchequer. Towards the close of the reign, it would seem that the King's surgeon was William Bradwardyn, who had the distinction of serving three sovereigns, Richard the Second, Henry the Fourth, and Henry the Fifth. He will be referred to again later.

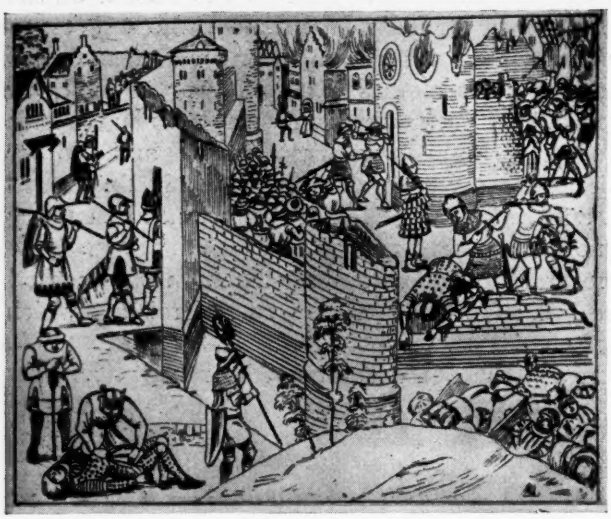

FIG. 368.—A French surgeon operating on the field of battle. (*Published in " Chirurgiens et Blessés ", by Cabanes.*)

Henry the Fourth, the son of John of Gaunt, who deposed Richard the Second, and became King of England, seems to have brought with him from France some French physicians, whom we may presume he came to know during his term of exile in that country. Their names, at any rate, sound foreign: LOUIS RECOUCHEZ or RECOUCHE, PETER DE ALBOCASSE, and DAVID DE NIGARILLIS. The first of these, Louis Recouche, was certainly a foreigner, because he received grant of denisation, or, as we should say to-day, papers of naturalization.[31]

Of the surgeons of Henry the Fourth we have but little to recount. There is one record, in 1410, of a grant for life to the King's servant, THOMAS

MORSTEDE, surgeon, of 40 pounds yearly.[32] This is no doubt the Thomas Morstede who is so well known as surgeon to Henry the Fifth (*Fig.* 369).

It is to be noted, too, that the surgeon's salary now is 40 pounds per annum, as against the 10 pounds granted to Master William in the reign of Henry the Third.

The other name which occurs on several occasions in the records of this reign is that of William Bradwardyn, or Bredewardyn. These records refer to him as esquire, surgeon, and serjeant-at-arms. It seems unlikely that there were two men of the same name, so it seems fair to assume that the two surgeons of Henry the Fourth were Thomas Morstede and William Bradwardyn, and that when Harry of Monmouth put on his father's crown and became King of England, he took over his two surgeons as well.

FIG. 369.—An imaginary picture by A. Forestier showing the departure for France of Morstede and a number of pressed surgeons. (*From the Collection of the Wellcome Historical Medical Museum.*)

Henry the Fifth began to reign on March 21, 1413, and he won the Battle of Agincourt on October 25, 1415. The details of the arrangements of the Expeditionary Force that he led are of considerable interest, for they concern the lives of the two notable surgeons—Thomas Morstede and William Bradwardyn. Preparations for the expedition were started a year ahead, and soldiers were enlisted under contract or 'indenture'. In this way, 2500 men-at-arms and 8000 archers were enrolled, and in addition numbers of non-combatants, such as carpenters, smiths, miners and gunners, armourers, yeomen of the pavilions, saddlers, physicians, surgeons, and chaplains.

The chief surgeon to the force was Thomas Morstede of London, and the indenture made with him is still preserved. He was to be ready to

attend a muster in May, 1415: he was to bring with him fifteen persons, of whom three should be archers and the others men of his own craft. His wages for the whole year were to be 40 ' marcs ' for himself and 20 ' marcs ' each for the other fifteen persons. The wages were to be paid in advance for the first quarter. It appears that the duty of organizing this fell to the service of the King's surgeon, who became, as we would style it to-day, Director General of the Army Medical Service.

In May, 1415, Morstede applied for money to purchase medical stores, as well as one cart and two horses to carry them.[33] Here is the germ of a medical service. To our modern ideas, the provision of one cart and two horses seems ridiculously inadequate to carry all the medical stores for an army preparing for a year's campaign. One may recall, however, that as late as 1808, at the beginning of the Peninsular War, only a little over one hundred years ago, when Sir Arthur Wellesley landed in Mondego Bay, the whole of the medical stores for the whole army was loaded into two carts drawn by bullocks. In assessing this campaign, therefore, one may conclude that it was the most carefully planned and the best organized and equipped expeditionary force that had ever sailed from this island. It throws a light, too, on the character of Henry the Fifth: he was not a hare-brained, devil-may-care adventurer, ready to risk his crown and kingdom on a mad expedition, but a rare general who took pains to leave as little as possible to chance.

The other surgeon who joined the expedition was William Bradwardyn. According to Sir H. Nicholas, he was in the retinue of the King, and had with him nine other surgeons.

The Physician to Henry the Fifth was named Nichol Colnet, and an indenture corresponding almost exactly with that of Thomas Morstede was made with him, and his wages were the same; but he was named alone and no other physician accompanied him.

There is not time here to follow the fortunes of the men engaged in this audacious but successful expedition. How, at the Siege of Harfleur, they had to deal with an outbreak of dysentery, in which 2000 men died and 5000 more had to be invalided home. Nor can we depict the hardships they endured in their march up the valley of the Somme, past Abbéville, Pont St. Rémy, Hangest, Corbie, Boves, Nesle, and Harbonierres. It was at Harbonierres that one of the soldiers, Bardolph, according to Shakespeare, stole a copper-gilt pyx from the church. The names of these towns are well known to many, and they are hallowed for all time by the graves of thousands of British soldiers who fell in the Great War.

Tired and lean from long marching, but with stout hearts, this little band of brothers met the French force on St. Crispin's Day, 1415, at Agincourt. " I would not, an I could, have here one single man more than I have," cried Henry.

Our three named doctors, Thomas Morstede, William Bradwardyn, and their medical colleague, Nicol Colnet, must have watched the battle with anxious eyes. I guess that they were stationed during the fight with the baggage animals, and alongside the chaplain who described the action. Their most notable patient would have been the Duke of Gloucester, who was wounded in the abdomen by a dagger thrust. He made a successful recovery.

What happened to the other wounded, one does not know. One suspects that those able to walk made their way back on foot, and that others were taken in country carts captured from the French. The English killed were put into a barn and there burnt. An exception was made with the Duke of York and the Earl of Suffolk, who were among the slain. Their bodies were boiled, so that their bones could be taken home for burial, the Duke of York at Fotheringay and the Earl of Suffolk at Ewelm.

After the death of Henry the Fifth, Morstede became surgeon to Henry the Sixth. He was a Sheriff of the City of London, and a supervisor of surgery, and is said to have written a goodly book on surgery ; but if he did, all trace of it is lost. He died in 1450, and was buried in the Church of St. Olave Upwell in the Jewry. His apprentice, Roger Brynard, inherited under his will his surgical instruments and ten marks sterling.

William Bradwardyn was a colleague of Morstede, though probably a little younger, for he was Vice-Master of the enfranchised art of Surgery in London when Morstede was Master, in 1422. So we can imagine that these two surgeons, who were present at the Battle of Agincourt, came back from the war, settled in the City of London, and occupied positions which corresponded in those days to the President and Vice-President of this College.

When Henry the Fifth died, the crown went to his infant son, Henry the Sixth, aged nine months. When he went to France to be crowned in Paris, it was felt necessary to send with him a surgical staff. This is a translation of the recommendation of the Council of the Kingdom :—

> May it please your Sovereign Majesty, of your special grace, and on the advice of your wise Council, to ordain that four surgeons take residence in your most honourable household, and to be obedient and answerable to William Stalworth, and to have each of them sixpence a day. And, in addition, to make a grant of twenty pounds sterling for sundry medicines, instruments, dressings (estuffes), and other necessaries necessary and pertaining to the office of Surgery for the use of you and the people of your household in this your present journey.[34]

The names of the four surgeons are not given, but this document is evidence for the presumption made earlier that the surgeon designated as Serjeant Surgeon was in fact the chief surgeon who was responsible for the actions of his colleagues and assistants, and also for the purchase and maintenance of drugs and instruments.

Stalworth evidently did his work well, for he was rewarded with the office of Steward of Cheillesmore, Co. Warwick, with the accustomed fees, wages, and profits.[35]

The next surgeon of whom we have records is Michael Belwell, a native of France, who was granted letters of denisation in 1443. No further facts have come to light about this man.[36]

At the age of 32 Henry the Sixth lapsed into a state of mental imbecility, a legacy perhaps from his French grandfather, and he became incapable of transacting the ordinary business of the realm. In this difficulty the Lords of the Council at Westminster advised the Lord Chancellor to appoint a Commission, consisting of Master John Arundel, John Faceby, and William Hatclyff, physicians, with Master Robert Wareyn and John

MARCHALL, surgeons, for the purpose of administering and attending to the person of the King. The powers given to these persons were indicated with great care. They were allowed to give the following medicines—electuaries, potions, waters, sirops, confections, laxative medicines, clysters, suppositories, gargles, baths, head shavings, and many others.[37]

Whether as the result of the activities of this Commission or not, the King recovered his reason, but only for a spell. In 1455 he relapsed into imbecility, and on this occasion the Dean of Salisbury, MASTER GILBERT KYMER, an "expert proved and notable man in the Craft of Medicine", was sent for to attend the King at Westminster.[38] This eminent physician and divine, at one time Chancellor of Oxford University, and author of *Dictarium de Sanitatis Custodia* (D.N.B.), was physician in the household of the King's uncle, Humphrey, Duke of Gloucester, at the time when he was called to attend his sovereign.

Further references to the King's surgeons are wanting, except that in the Regulations for the Household of Henry the Sixth for the year 1454, Robert Waren is cited as serjeant surgeon, John Marchall as yeoman-surgeon, and THOMAS BEKBANK as groom-surgeon.[39]

In 1461 the imbecile King Henry was deposed, and Edward the Fourth put in his place. One of the acts of the first year of his reign was to make a grant of forty pounds a year for life to MASTER JAMES FRIIS (or Frus, or Fryse), his physician. This man was born in the parts of Friesland; he remained in the service of his royal master for twenty years, and received several grants of land and money.[40]

In the second year of his reign the King appointed as his surgeon, MASTER WILLIAM HOBBYS, and granted to him some property, including a wharf and crane in the parish of St. Martin in the Vintry, London. In another grant, made eight years later, Hobbys is described as the principal surgeon of the body.[41] Master William Hobbys was evidently a man held in high esteem in the City of London, for in 1461 he was Warden of the Company of Barber Surgeons, and he survived in the troublous times in which he lived to become the body surgeon of Richard the Third.

In 1475, at the head of an army, Edward the Fourth invaded France, but he accomplished little, owing to the astuteness of Louis the Eleventh. There is a picturesque account of the meeting of these two kings on the bridge at Picquigny on the Somme, a town well known to many members of the British Expeditionary Force during the Great War. The medical arrangements for this expedition are preserved in Foedera under the heading of "Wages for the Voyage to France", and so we are able to furnish the actual names and wages of those that served.

Master Jacob Fryse was the physician, receiving as wages two shillings a day. Master William Hobbys, physician and surgeon to the body of the King, had eighteenpence a day, and one surgical assistant at sixpence a day. Then there were seven other surgeons who received one shilling, and five who had only sixpence. The total wages for the medical services for the quarter came to £65 19s. 6d.[42]

Some at least of these men were of high standing in their profession, for four of them, namely, RICHARD BRIGHTMAN, THOMAS COLLARD, RICHARD

CHAMBERS, and SIMON COLE, at one time held the office of Warden to the Company of Barber Surgeons.[43]

There are very few records of any sort of the brief reign of the unfortunate King Edward the Fifth, and as far as can be ascertained there is no mention of any medical men.

FIG. 370.—An army surgeon extracting an arrow on the field. Sixteenth century.

FIG. 371.—An army surgeon. Sixteenth century. Note the pointed instrument case near the surgeon's right foot.

Neither is the brief and stormy reign of Richard the Third productive of information concerning members of our profession. There is but one item of information to record, namely, that Richard seems to have continued in office his predecessor's medical attendant, namely, William Hobbys, and granted to him 40 pounds yearly for life.[44] The name of this surgeon disappears from the records in 1483, and there is naught to show what manner of man he was, nor whether he was near his royal master on fatal Bosworth Field.

It is surprising that the reign of Henry the Seventh is not more productive in records of surgeons—in fact it is very scanty. So far only one surgeon has been discovered,

FIG. 372.—A surgeon's tent near the battle. Note the instrument case hanging in the tent. (*Figs. 370–372 are from "Der Arzt und die Heilkunst", by Herman Peters, Leipzig, 1900, by kind permission of Eugen Diederichs Verlag.*)

namely, WILLIAM ALTOFTES of Atherston, and of him all we know is that in 1487 he is described as "principal surgeon of the king's body", and that he was given a grant of 40 marks a year for life.[45] It has been discovered, too, that he was alive in 1520, in the reign of Henry the Eighth, for there is a notice in that year that he was paid his annuity.[46]

As would be expected from a man of his character, Henry the Eighth appointed his own surgeons, and his first was MARCELLUS DE LA MORE, who, in 1510, was described as the King's surgeon and was granted 40 marks yearly, during pleasure. He seems to have proved satisfactory, for three years later, in 1513, he was made "serjeant of the King's surgeons, with wages and precedence as customary since the reign of Edward the Fourth". More must have been closely associated with the King's physician, DR. THOMAS LINACRE, the founder of the College of Physicians. One likes to think that the relations between the physicians and surgeons were as friendly as they are now, and that more received an invitation to be present at the ceremony of the foundation.

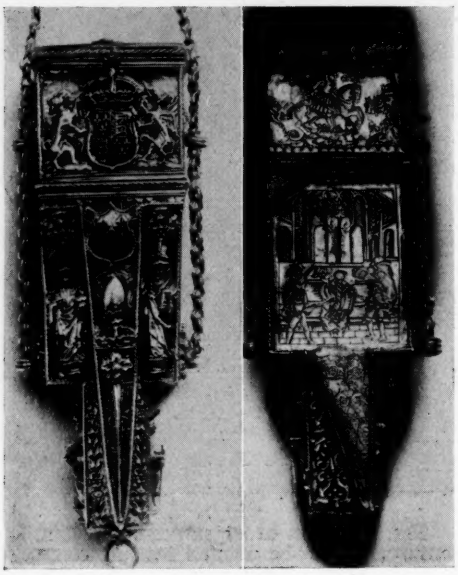

FIG. 373.—A silver instrument case bearing the royal Tudor arms and the arms of the Barbers Company. It may have belonged to Thomas Vicary. (*In the possession of Messrs. Creighton Bros.*)

The fact that Marcellus de la More is cited as serjeant of the King's surgeons implies naturally that there were others, and a search in the accounts of the royal household reveals that in 1516 there was a certain JOHN VERARY, who is described as chief surgeon at a salary of £61 13s. 4d. for life, and in 1509 there is a brief notice of a payment of £61 13s. 4d. to Mr. JOHN, the King's surgeon.[47]

In 1519 Henry seems to have appointed another surgeon in the person of ANTHONY CHABO, a surgeon of London, with an annuity of 20 pounds, and he retained his office at least as long as 1523.[48]

THOMAS VICARY (*Fig.* 374).—The direct successor to Marcellus de la More in the office of serjeant surgeon was Thomas Vicary. The following are the terms in which his appointment is described :—

> THO. VICARIE.—To be serjeant of the King's surgeons, and chief surgeon to the King, with allowances both "le bouge the Courte" of the King's household, and of wine, etc., for cures ; which office of serjeant was granted by patent 6 Aug. 5 Henry 8. to Marcellus de la More, chief surgeon to the King. Also grant, in reversion, to the said Tho. of the annuity of 40 marks which was granted to the said Marcellus by patent 3 No. 7 Henry 8., to hold from the time when the said office shall be vacant.

It is possible now to trace a direct sequence of these Court Physicians and Surgeons from the time of Edward the Confessor to Thomas Vicary. We

have seen how in early times they were dignitaries of the Church, how later they became secular and gradually more and more specialized. We have seen the important part they took in the medical arrangements for war; how their duties and privileges, at first vague, were defined. Though they

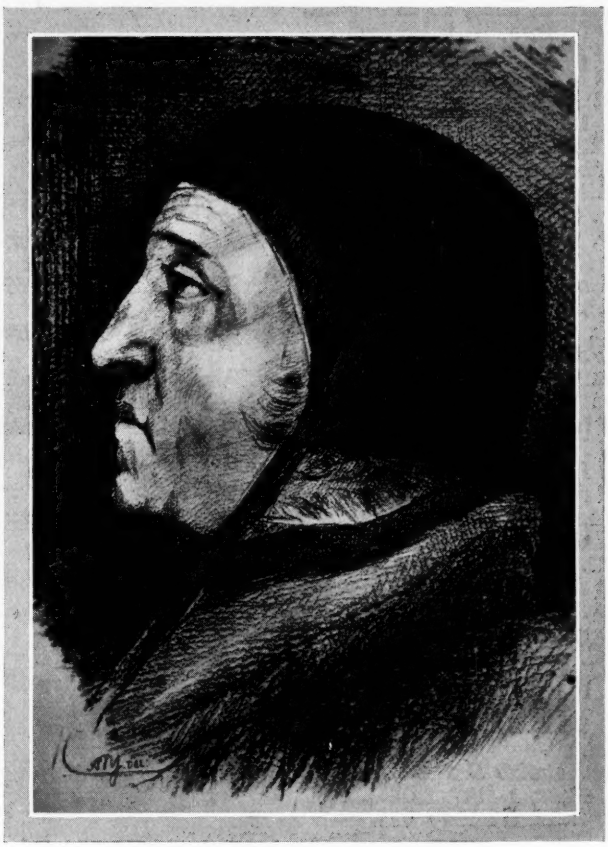

Fig. 374.—Thomas Vicary.

left no writings behind them, they were the leading men of their time and we should not lose sight of them. In honouring their names, we honour this College, whose aim it is to preserve the best of those traditions which have been handed down to us.

Grateful acknowledgements are made to Sir D'Arcy Power, R. R. James, Esq., C. J. S. Thompson, Esq., Messrs. Creighton Bros., and particularly to the Staff of the London Library for their unfailing courtesy and co-operation.

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THE TREATMENT OF GLIOMATA AND PITUITARY TUMOURS WITH RADIUM.

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Of all malignant tumours the gliomata would, *a priori*, seem to be the most favourable for radium treatment. They do not infiltrate tissues other than those of the central nervous system itself, and remain, almost without exception, within the confines of the meninges; they give rise to no metastases; they do not invade lymphatic glands. Further, they are, for the most part, composed of cells of a primitive character.

It is, perhaps, convenient for the purposes of this paper to adopt some of the terms employed by Bailey and Cushing[1] in their attempt to classify the gliomata, as some of them have already, though perhaps prematurely, passed almost into general use. We recognize, however, that that classification, and indeed some of the terms, must at present be only provisional, just as are certain other names given to the same tumours by previous workers, notably Greenfield.[2] The types of glioma which we have so far had the opportunity of treating with radium are, according to the nomenclature of Bailey and Cushing, spongioblastoma, oligodendroglioma, medulloblastoma, and astrocytoma. It is to Dr. Greenfield that we are indebted for all histological examinations and opinions, without which this paper would have little value.

Before we undertook the observations upon which the paper is based, one of us had employed radium in a number of cases of glioma, quite empirically, and with little to guide either the mode of application or the dosage. Nevertheless the results were such as to justify the general conclusion that under the influence of radium gliomata were apt to behave in an unusual manner, in some cases unexpectedly to recede, remain inactive, or even apparently to disappear. One example will suffice to support this generalization.

A professional man, 47 years of age, came under the care of Dr. Gordon Holmes with a history of cerebellar symptoms extending over three months. In September, 1921, the cerebellum was exposed, and a mass of soft, gelatinous tumour was removed from within the right hemisphere. He made a good recovery, but symptoms recurred three months later, and a second operation was performed. Masses of growth were removed, and 62 mgrm. of radium, screened with 2 mm. of lead, introduced into the middle of the growth. The radium was left in position forty-eight hours. The total dose given was 2976 mgrm.-hours (Dr. Martin Berry). After a stormy convalescence the patient made an excellent recovery, and remains well and almost free from symptoms after nine years.

Histology.—This is a very cellular tumour, with cells of two distinct types. Some are large with abundant cytoplasm and indefinite processes passing some

distance from the cell body. In some cases the processes of one cell seem to fuse with those of another, but this is not very clear. The nucleus of these cells is large, vesicular, and palely staining, with a central or paracentral nucleolus. The other cells are small, with round, dark-staining nuclei and very little cytoplasm. The blood-vessels are numerous, with fairly thick walls, usually showing endothelial hyperplasia. Mitoses are numerous in some parts. The tumour tissue removed at the two operations has identical characters. The large cells with clear nuclei may be neuroblasts. If so, the tumour would correspond with Bailey and Cushing's ' medulloblastoma neuromatosum '.

We are aware, of course, that quite apart from radiation treatment some patients with gliomata survive for many years, and that some types of glioma have at least a very low degree of malignancy. But the case quoted is one, not of survival, but of apparent cure with partial restoration of cerebellar function.

PRINCIPLES OF IRRADIATION.

The treatment of cerebral tumours by radium is based upon the same principles as guide the clinician in all radium therapy. The fundamental element is the variation in the response between normal neuroglial tissues and that of tumours. Both from the study of clinical cases and from experimental irradiation of animals' brains, it is evident that nerve tissue is resistant to the effect of gamma rays to such a degree that within therapeutic dosage no caustic effect is obtained. The effect on the tumour cells is inhibitory to growth, and the combination of sensitivity of the neoplasm and resistance of normal brain offers a field of therapy well worth exploring. It is not denied that destruction of tissue in the immediate vicinity of the source of irradiation can be produced experimentally[3]; necrosis so obtained is due to inadequate filtration and the effects of beta particles, or to an excessive intensity of gamma irradiation.

In our own experiments necrosis has been obtained by using radon seeds of 3- or 5-mc. strength screened by 0·3 mm. of platinum or silver or by insertion of a 10-mgrm. tube left in position for ten days. The experimental evidence was obtained by the introduction of radon seeds and radium needles into the cerebral hemispheres of rabbits. The effect of variations in screenage, dosage, and time were studied. The rabbits were killed at varying intervals —from a fortnight to five months—after removal of the radium. In some cases the radon seeds were left *in situ* permanently. These experiments showed that necrosis occurred in the immediate vicinity of the source of irradiation and it did not extend beyond a few millimetres from it; necrosis was directly proportional to the dosage and inversely proportional to the screenage. It is obvious that some of the changes were due to the trauma of the needling, as we know that they can be produced by the use of dummy needles or empty seeds. The effect of trauma can be entirely eliminated by placing the radium between dura and bone, or better still by employing surface irradiation. After recovery from operation the animals appeared to be normal in every respect. A full account of these experiments will be published later. The experiments go to prove that irradiation of the brain requires as much care, thought, and attention to details as irradiation elsewhere. The success of treatment depends primarily upon the histological nature of the

tumour, and secondly upon accurate and correct irradiation. The first condition is at present beyond clinical control, although methods of increasing the radiosensitivity of tumours are being tried, and not without some encouragement. The second condition depends upon technique of irradiation. Reduced to its principles, it depends upon the linear intensity, the screenage, and the time factor. A low linear intensity, pure gamma irradiation, and a prolonged time factor, give the patient a greater chance of recovery.

CHOICE OF METHOD OF IRRADIATION.

The two methods at the disposal of the surgeon are interstitial and surface irradiation. The choice of the method depends upon two factors: (1) The localization of the tumour; (2) The operability. Tumours situated in the pituitary region are not suitable for surface radium therapy. Unlocalized tumours, or those situated near the base of the skull, the mid-brain or near the medulla, are not suitable for interstitial irradiation. The ideal, however, is to submit each case to a two-stage treatment combining interstitial irradiation with surface treatment whenever possible, and employing high-voltage X-ray therapy as a second stage in cases when surface therapy is not practicable.

Decompression. — Preliminary decompression is an essential step of the treatment. It offers relief from increased intracranial tension, whilst a window is created through which the underlying tumour can be irradiated without the screenage offered by bone, an appreciable factor in some skulls; the risk of radium osteo-necrosis is eliminated; removal of the tumour becomes possible in some cases, and even partial removal of a tumour is of definite benefit; a biopsy is obtained; and interstitial irradiation can be carried out.

INTERSTITIAL IRRADIATION.

Needles.—The use of needles containing small quantities of radium (1 to 2 mgrm. per needle) is preferred to the use of radon seeds in all situations with the exception of the pituitary. Needles of 27 or 33 mm. over-all length are employed; accurate distribution of the needles within and around the the neoplasm is possible (*see Figs.* 383–385); to obtain such distribution with seeds is a feat rarely accomplished. The falling intensity of emanation is a factor of equal importance, and in the treatment of gliomata it is a factor which cannot be overestimated. The needles, threaded with carbolized silk, are inserted equidistally 1 cm. apart, great care being taken to avoid blood-vessels. All the threads are tied together and cut short; the knot is placed between the dura and the flap, only one thread being brought out through the incision. The wound is closed without drainage. The needles are left in position for seven days, the wound is then partially re-opened, and with the single thread as a guide the knot is found and the needles are removed. An alternative method used occasionally is to employ silver wire instead of thread and to bring all the wires outside the incision (*see Fig.* 386).

Seeds.—The use of seeds is indicated in the pituitary region (*see Figs.* 389–392). Very great caution must be exercised in the irradiation of this area; two or three seeds each containing 1·2 mc. screened by 0·5 mm. of

platinum are inserted. The seeds tend to fall to the bottom of the sella, and unless a great many seeds are used, irradiation is not evenly distributed. The possibility of producing permanent blindness by excessive dosage must be kept in mind. In connection with the dosage of radon which may be used in pituitary tumours, we have been given the opportunity of making use of the following case :—

M. S., female, age 25, was first seen at St. Thomas's Hospital in July, 1928, with bitemporal hemianopia and headaches of eighteen months' duration. She was operated upon in October, 1928, when, through a transfrontal osteoplastic approach, a quantity of growth was removed from a pituitary tumour, which proved

FIG. 375.—*Case* M.S. Illustrating ten seeds in the region of the optic chiasma.

to be a so-called adenoma (adenomatous hyperplasia). At the same time ten radon seeds were inserted into the cavity ; these were subsequently found radiographically to be aggregated into a single mass close to the optic chiasma (*Fig.* 375). The visual fields improved to a slight, but quite definite, extent for five months, but ten months after the operation they were found to have deteriorated again. The discs were found to be atrophic, and there were no hæmorrhages. Accordingly, in September, 1929, a second operation was performed. The original flap was again turned down, and the left frontal lobe elevated so as to expose the sellar region. There was no increased intracranial pressure. The seeds, together with a " mass of white tissue resembling slough ", were removed. This tissue showed " excessive glial proliferation ; thrombosis ; no neoplastic tissue ". The patient made a good recovery in every way except as regards vision, which steadily deteriorated until complete blindness resulted (*Figs.* 376–379).

This case is of the greatest interest from the point of view of the number of seeds to be employed, and the difficulty of spacing them in the soft, or even cystic, masses which constitute the majority of pituitary tumours. We ourselves have never employed more than four seeds, each with 1·5 mc. screened by 0·5 mm. of platinum or gold. Here ten seeds were used, each of

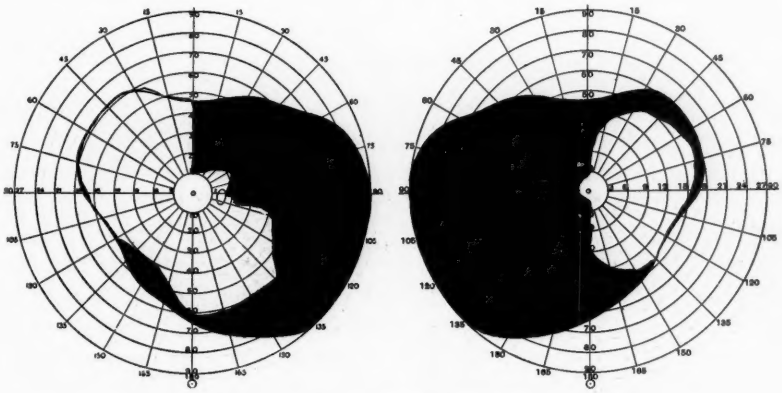

FIG. 376.—*Case* M.S. Visual fields before operation.

2 mc. screened by 0·3 mm. of platinum, and they were aggregated close to the optic chiasma. In spite of this the vision improved for some months before commencing the deterioration which ended in blindness (*see Figs.* 376–379). We are not prepared to say whether this was a late result of placing what we consider to be a non-therapeutic dose of radon close to the optic

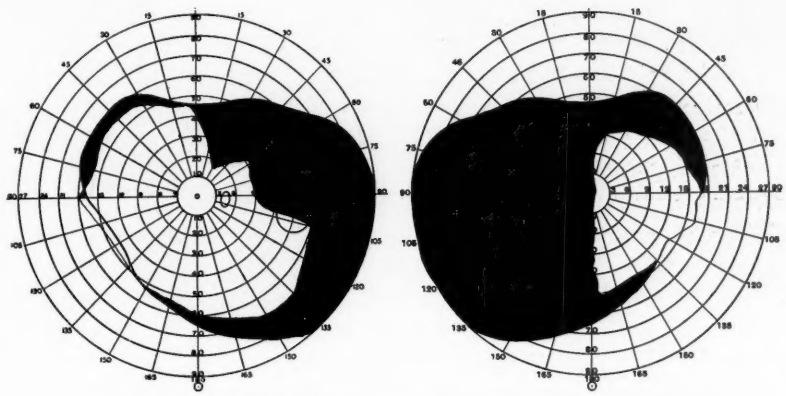

FIG. 377.—*Case* M.S. One month after insertion of radium.

chiasma, or an indirect effect upon the visual paths by causing the glial proliferation noted later in their neighbourhood. Possibly neither of these suggestions embodies the true explanation, which might equally well be that the left eye had already been so badly damaged before the first operation that eventual recovery of vision was impossible; whilst the adenomatous mass may still have been encroaching upon the right side of the chiasma, the

operation having been done upon the left side. It is our experience that the vision improves most upon the side operated upon, and it is our practice, therefore, always to operate upon the side of the better eye.

It is evident from the case reported above that some effort had to be made to ensure an even distribution of irradiation over a wider area and giving more

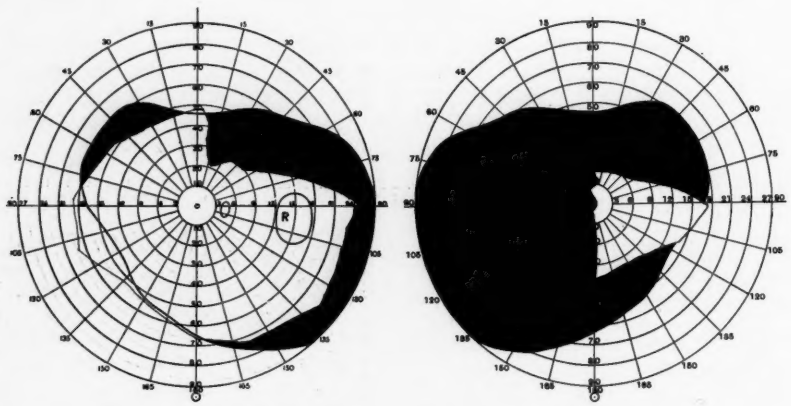

FIG. 378.—*Case* M.S. Five months after insertion of radium.

control as to the position of the radio-active focus. To secure this object a special container was made for us by Dr. Alton, of the London Radium Institute. Its object is to provide a source of irradiation filling the sella turcica and projecting upwards into the suprasellar space. It is made of silver (1 mm.), is oblong, and is filled with the desired amount of radon; 8, 10,

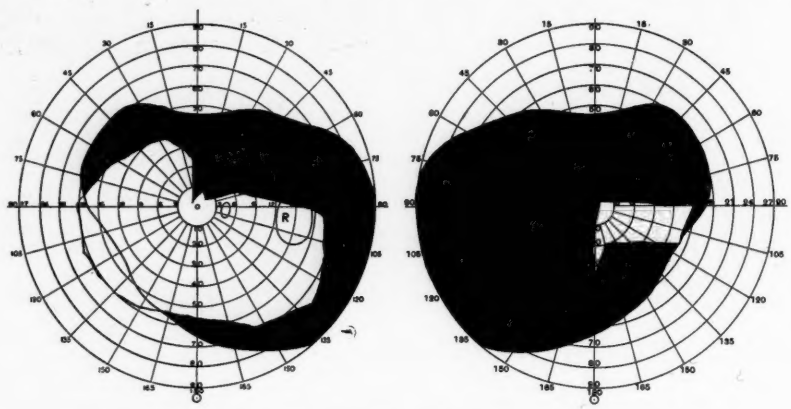

FIG. 379.—*Case* M.S. Eight months after insertion of radium.

or even 15 mc. can be used. The peripheral area irradiated is four to six times as wide as that irradiated by two or three separate seeds (*see Figs.* 393, 394).

For interstitial irradiation of tumours of the cerebellum seeds present certain mechanical advantages over needles (*see Fig.* 387).

Immediate Effects of Interstitial Irradiation.—Healing of the wound is slower than normal; leakage of cerebrospinal fluid may continue for several

weeks. If radium is placed above the tentorium, there are no untoward symptoms of any importance, though papillœdema is as a rule increased and retinal hæmorrhages may occur, though not so commonly as with surface treatment. Below the tentorium, interstitial irradiation is not so well tolerated; headache is very common, persistent hiccup has been noted in several cases, polyuria is the rule, and escape of cerebrospinal fluid may be continuous.

SURFACE IRRADIATION.

The method employed is that of a skull-cap of 'sorbo' rubber, or 'columbia paste' (*Figs.* 380, 381). It is applied as soon as the operation wound is soundly healed. The radium-skin distance is 15 mm. The quantity of radium varies from 50 to 75 mgrm. in numerous needles. The area treated is wide, half the head (right or left side), or the occipital or frontal areas, according to localization. With this quantity of radium it is possible to obtain an inhibitory effect on tumours situated near the corpus callosum, in the lateral ventricles, or encroaching on the mesial aspect of the cerebral hemisphere. That a cap so constructed, carrying 50 mgrm. of radium, emits irradiation reaching a depth of 4 or 6 cm. has been proved experimentally by Dr. W. G. Evans, who, working in conjunction with one of us, carried out the following experiment: An accurately fitting cap was made and applied to a cadaver after sagittal bisection of the head and insertion of an X-ray film between the two hemispheres. At the end of four hours the film when developed showed obvious evidence of having been submitted to irradiation. These experiments will be published in full elsewhere.

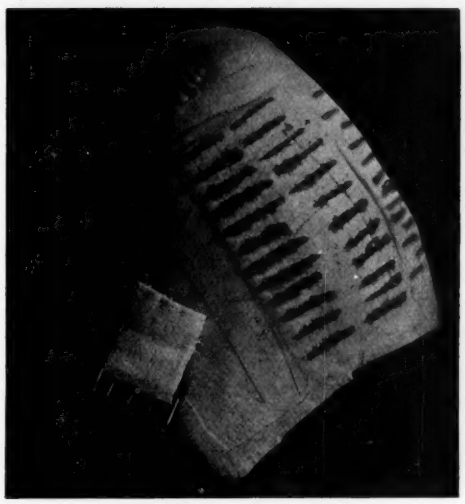

FIG. 380.—'Sorbo' rubber cap for treatment of a tumour in the right fronto-parietal region, and showing the distribution of the needles.

By this method a large area of the brain can be treated. The treatment is intermittent, fourteen to eighteen hours being given daily for a period of from two to three weeks in cases where a previous interstitial treatment has been given, and from four to six weeks if surface application is the sole method employed.

Immediate Effects of Surface Irradiation.—At first the papillœdema is increased, but after seven to ten days this diminishes. Retinal hæmorrhages have occurred in so many cases that they are now looked for and expected; but their significance is not yet understood.

The effect on the secretion of cerebrospinal fluid is interesting. It is known that X-radiation to the head decreases the activity of the choroid plexus; there is evidence that a similar effect is obtained by radium, but

further study is necessary to obtain definite evidence. After operations upon the brain there is an increase in the production of cerebrospinal fluid. This may be due to hypersecretion resulting from the manipulation, and so be similar to the increase which can be produced experimentally by the administration of brain extract. The increase at times is so great that repeated

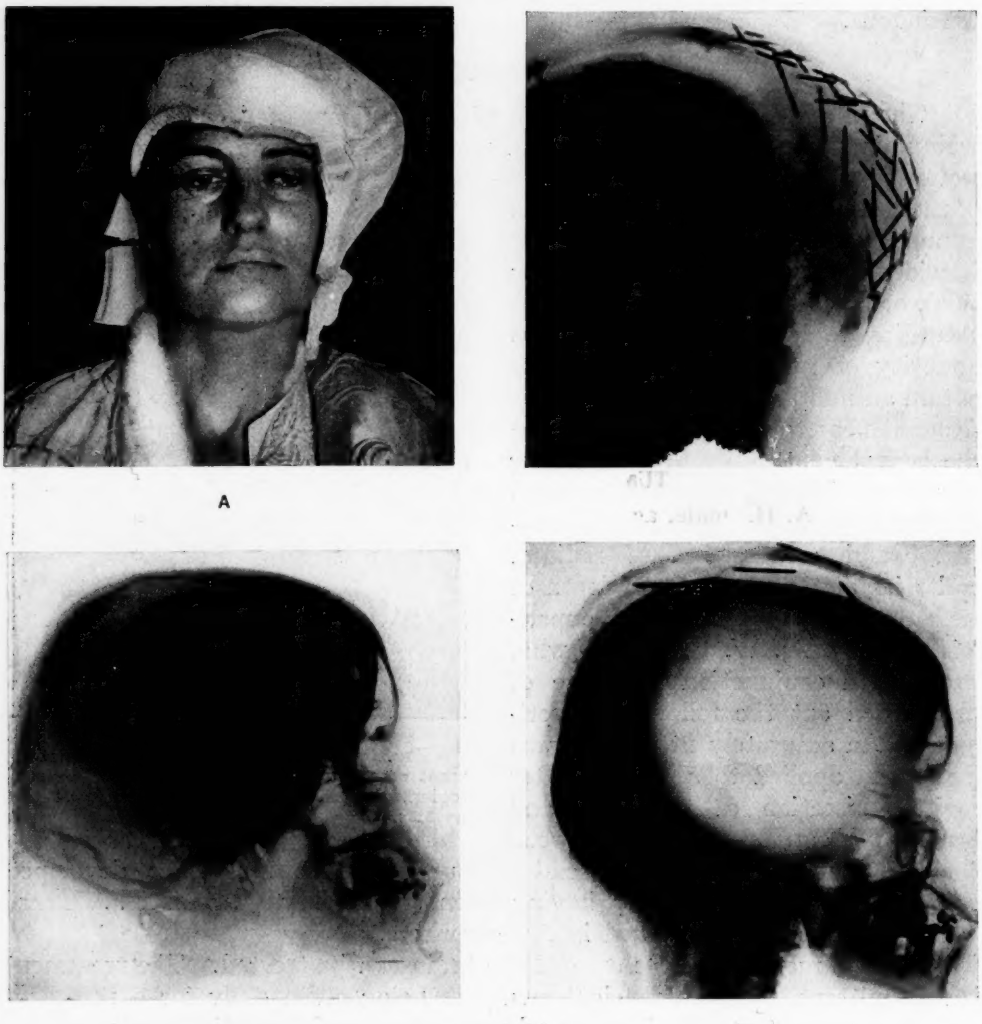

FIG. 381.—A, 'Sorbo' rubber cap for surface treatment of tumour in the temporal region. B, Skiagram showing the distribution of the needles in relation to the tumour and the skull. C, Skiagram showing portion of bone removed at operation. D, The same, illustrating the area under the influence of the radium; the clear area is produced by the radium and corresponds to the position of the bony opening as shown in C.

tapping or lumbar puncture is necessary. In the majority of cases there is a gradual diminution of the size of the swelling, and in all successful cases the area of the flap becomes concave. Surface irradiation is continued until all the hair is shed, and in some cases until peeling of the scalp results. In the latter event the regeneration of hair follicles does not take place.

Dosage.—The criticisms of the 'milligram-hour' method of notation are numerous and in some ways justifiable, but the alternative methods 'erg-cubic-centimeter milligram-hour' of John Murdoch or 'skin erythema dose' of Quick, Failla, and Quimby are not universally accepted. All methods of notation are faulty, but until such a time as a universal unit of measurement is available, we prefer to note the dose in 'milligram-hours', giving the necessary data of number of foci, amount of radium, time, screenage, distance, and method of irradiation.

CASE REPORTS.

The following twenty-one cases were selected to illustrate the type of tumour treated and the methods employed. They do not represent any particular histological type, but rather a mixed group illustrating failures and successes, pointing out the dangers of the treatment, the disappointing end-results in some, and apparent successes in others. A series of cases treated by surface irradiation by one of us is not included, although the results are most encouraging, as the study of that series is not yet completed. The few cases treated by teletherapy (the so-called radium 'bomb') are also omitted, as it is not desirable at present to express any opinion on this method of radium therapy.

TUMOURS OF THE CEREBRUM.

Case 1.—[illegible], aged 37 years. (Dr. J. P. Martin.)

FIG. 382.—*Case* 1. Oligodendroglioma showing calcification in the frontal and parietal area.

HISTORY.—Two years. Frontal headache; vomiting; ten attacks of loss of consciousness; blurring of nasal edges of both discs.

X RAY (*Fig.* 382).—Definite irregular opacity in left fronto-parietal region.

OPERATION (Sept. 27, 1929).—Left parieto-temporal opening, and large subcortical tumour exposed. The tumour was found to be deeply infiltrating the brain; the main mass of the tumour, which was the size of a small tangerine orange, was removed, but the deepest part was not reached. The bone-flap was removed and the skin closed.

HISTOLOGY.—Oligodendroglioma.

RADIUM TREATMENT (Nov. 8).—Eighteen needles each containing 1·3 mgrm. of radium element, screened by 0·6 mm. of platinum, were inserted into the periphery

FIG. 383.—*Case* 1. Showing decompression and eighteen radium needles in position.

of the tumour (*Fig.* 383) and left *in situ* for 7 days. Total, 3721 mgrm.-hrs. This was followed by surface application of 80 mgrm. of radium at a distance of 15 mm. by means of a columbia-paste cap, 16 hours daily for 10 days. Total, 12,960 mgrm.-hrs. There was marked erythema and the hair rapidly fell out.

AFTER-HISTORY.—The patient returned to work and remained well and active for one year; he died suddenly in November, 1930, but no details could be obtained.

Case 2.—K. H., female, age 49 years. (Dr. Gordon Holmes.)

HISTORY.—One and a half years. Attacks of momentary faintness. Recently loss of consciousness. Three weeks' pain in the eyes and forehead; vomiting. Persistent frontal headaches. Bilateral papillœdema; left internal strabismus.

OPERATION (Nov. 8, 1929).—Left temporo-parietal decompression. Subcortical tumour removed from the posterior end of the second temporal gyrus. The tumour was red, soft, and the size of a hen's egg.

RADIUM TREATMENT.—Seven days after operation 7 needles each containing 2 mgrm. of radium, and 3 needles each containing 1·3 mgrm., were inserted into and around the site of the tumour interstitially. The radium was left in position for 12 days. Total, 5184 mgrm.-hrs. On Jan. 10, 1930, a skull-cap made of columbia paste carrying 35 mgrm. of radium was applied for 12 hours daily for 20 days. Total, 10,900 mgrm.-hrs.

HISTOLOGY.—Glioma of rather unusual structure owing to the number and

large size of rosette arrangements, in which elongated cells are grouped with their nuclei arranged almost in palisade formation, and a long process extending inwards into a non-nucleated area. Cells chiefly bipolar. Mitotic figures common.

AFTER-HISTORY.—Three months later the patient was clinically improved, but the hernia cerebri was very tense. She died from the disease five months after treatment.

Case 3.—J. B., female, age 13 years. (Dr. Kinnier Wilson.)

HISTORY.—Ten months. Astereognosis in left limbs; papillœdema; left homonymous hemianopia.

X RAY.—Showed a tumour, calcified in parts, in the right occipital area.

OPERATION (Dec. 7, 1929).—Right-sided osteoplastic flap. Tumour removed from occipital lobe.

RADIUM TREATMENT.—Thirteen needles were inserted—9 needles each containing 1·3 mgrm. of radium, screened by 0·6 mm. of platinum, into adjacent brain

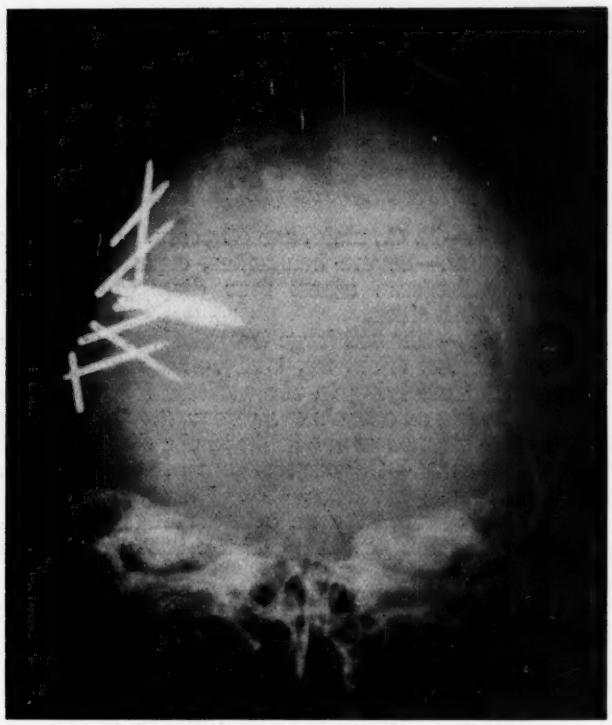

FIG. 384.—*Case* 3. Antero-posterior view showing thirteen needles in position. Of these, nine are around the periphery and four in the centre of the space previously occupied by the tumour.

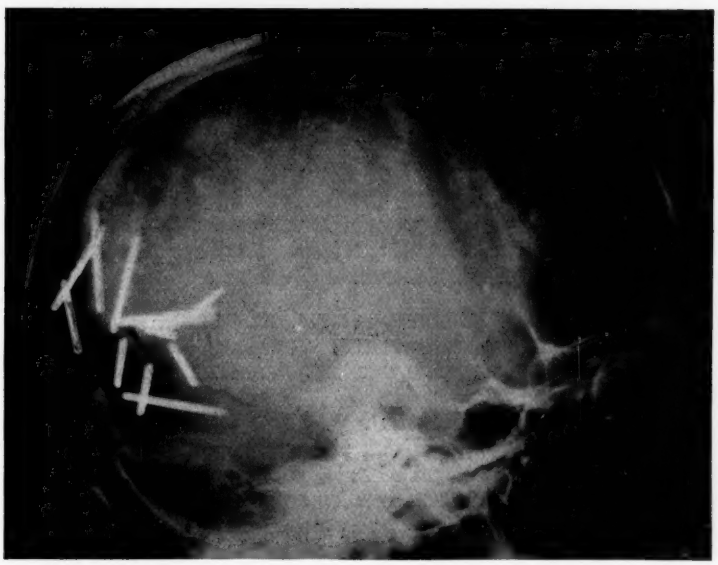

FIG. 385.—*Case* 3. Lateral view, showing radium needles in position.

posteriorly ; 4 needles each containing 2 mgrm., screened by 0·65 mm. of platinum, into the space previously occupied by the tumour (*Figs.* 384, 385). The needles were left in position for 4½ days. Total amount of radium used, 20 mgrm.; period of irradiation 96 hours. Total interstitially, 1917 mgrm.-hrs. Surface irradiation by means of a columbia-paste cap on Jan. 14, 1930. Total quantity of radium used, 70 mgrm., screened by ½ mm. of platinum and ½ mm. of brass. Daily application 8 hours for 12 days, with occasional interruptions in the treatment. Total, 5180 mgrm.-hrs. The hair came out and there was definite erythema.

HISTOLOGY.—Oligodendroglioma.

AFTER-HISTORY.—The patient remains well to date.

Case 4.—E. C., male, age 43 years. (Dr. Hinds Howell.)

HISTORY.—Severe headaches, especially at the back of the head. Unsteady gait. Papillœdema about four dioptres. Nystagmus. Motor power good, and equal on both sides.

X RAY.—Showed large irregularly-shaped tumour in the left parietal region.

OPERATION (Feb. 28, 1930).—Left parietal flap. Discoloured tumour in the brain substance lying in the parietal lobe behind the post-central gyrus. Mass of tumour as big as a hen's egg removed. The tumour had a pedicle which passed far into the substance of the brain in the direction of the corpus callosum.

HISTOLOGY.—Oligodendroglioma.

RADIUM TREATMENT.—Six days after operation 10 needles each containing 1·33 mgrm. of radium, 27 mm. over-all length, screened by 0·6 mm. of platinum, were inserted into the periphery of the tumour and left in position for 7 days. Total, 2226 mgrm.-hrs.

AFTER-HISTORY.—The patient remained well for ten months. He was then re-admitted to hospital with a tense hernia cerebri and recrudescence of symptoms. He is at present under treatment.

Case 5.—E. K., female, age 39 years. (Dr. Gordon Holmes.)

HISTORY.—The history is difficult to get and is incomplete. Five months' vomiting and three months' headache. Inability to fix her attention. Drowsy. No fits of any kind. Diminution of sensation on the left side.

X RAY.—Skiagram of head negative.

OPERATION (Jan. 31, 1930).—Large right parietal flap. Dark-red infiltrating tumour on the lower lateral surface of the frontal lobe reaching the surface above and in front of the Sylvian point, infiltrating the brain extensively. Much tumour tissue was removed with very little loss of blood.

RADIUM TREATMENT.—Twelve mgrm. of radium were inserted at the periphery and 6 mgrm. into the cavity previously occupied by the tumour. Total, 19·3 mgrm.

AFTER-HISTORY.—The patient died in coma forty-eight hours after operation. Death from cardiac failure.

POST-MORTEM.—The anterior end of the corpus callosum appeared to be infiltrated with tumour, and farther back the infiltration reached the caudate nucleus. Histologically the tumour is a glioma which varies very much in structure, many cells being collected in clusters or small rosettes. In other places the cells are loosely scattered in a matrix of neuroglial fibres.

Case 6.—J. P., male, age 13 years. (Dr. Gordon Holmes.)

HISTORY.—Four months. Headaches, vomiting, increased irritability; no loss of power or wasting. Reflexes normal. Weakness of right external rectus. Slight weakness of left hand.

X RAY.—No abnormality detected.

OPERATION (April 8, 1930).—Right temporal decompression. Removal of a large tumour from the right temporal lobe.

RADIUM TREATMENT.—Fourteen needles each containing 1·3 mgrm. of radium were inserted at the periphery of the cavity previously occupied by the tumour, and 10 mgrm. into the cavity.

AFTER-HISTORY.—The patient died thirty-six hours after operation.

HISTOLOGY.—Glioblastoma.

Case 7.—M. S., female, age 24 years. (Dr. Adie.)

HISTORY.—Four months. Headaches, vomiting, dimness of vision; amenorrhœa for three months. Bilateral papillœdema of about three dioptres in each eye, with some hæmorrhages. Left pupil larger than right. Muscular power and tone good. Diminished sensation to pin-prick on left side of body. The patient has had four attacks of unconsciousness associated with convulsive movements of jaw and clonic contractions of arms.

X RAY.—No evidence of any abnormality.

OPERATION (Feb. 10, 1930).—Right temporal decompression (Mr. Donald Armour). Subcortical tumour the size of a large oyster removed from the temporal area. A month after operation there was no diminution of papillœdema.

HISTOLOGY.—Astrocytoma fibrillare.

RADIUM TREATMENT.—A columbia-paste cap carrying 55 mgrm. of radium in 34 needles was applied at a distance of 15 mm. from the skin. The patient received 18 hours daily, and treatment continued for 21 days. Total, 20,590 mgrm.-hrs. Total loss of hair on the side treated and over a small area on the opposite side. The skin peeled on the side treated, papillœdema disappeared, and the general condition of the patient improved. There were no fits.

AFTER-HISTORY.—The hernia cerebri has entirely disappeared; the operation area is concave; there is no papillœdema; menstruation has returned. She has had three fits since leaving hospital.

Case 8.—G. A., female, 34 years. (Sir James Purves-Stewart.)

HISTORY.—Thirteen months. Headaches and vomiting; momentary blindness; deterioration of vision; papillœdema; dragging of right leg. Fits.

X RAY.—Pneumoradiogram showed both lateral ventricles displaced to the right.

OPERATION.—Left frontal decompression (Mr. Arthur Evans). Tumour not removed. Following operation: aphasia, right-sided weakness, incontinence, and marked hernia cerebri.

RADIUM TREATMENT.—Columbia-paste plaque applied: 50 mgrm. of radium. Continuous application for 24 hours followed by 12 hours daily for 10 days. Total, 6600 mgrm.-hrs. Peeling of skin over forehead and loss of hair.

AFTER-HISTORY.—Complete recovery with the exception that vision in the left eye is reduced to perception of light. Well and symptomless three and a half years later.

Case 9.—W. W., male, age 61 years. (Sir James Purves-Stewart.)

HISTORY.—Right homonymous hemianopia; hemi-anæsthesia; right ankle-clonus; Jacksonian fits involving right upper and lower limbs.

OPERATION (March 12, 1926).—Left parieto-occipital osteoplastic flap (Mr. Arthur Evans). Subcortical tumour present in left occipital lobe.

HISTOLOGY.—Gliosarcoma.

RADIUM TREATMENT (March 25, 1926).—Columbia-paste cap 21·85 mgrm. applied 16 hours daily for 16 days. Total, 5632 mgrm.-hrs. The patient recovered and remained well till November, 1926, when he had several fits. A second radium treatment was given, 50 mgrm., 14 hours daily for 15 days, then the amount was reduced to 20 mgrm. for a further 6 days. Total, 14,700 mgrm.-hrs.

AFTER-HISTORY.—The patient remained well for twelve months, then died after a fit.

Case 10.—A. W., male, age 9. (Dr. Gordon Holmes.)

HISTORY.—Five years' history of fits recently getting more frequent and worse; for three years associated with loss of consciousness.

OPERATION (May 10, 1930).—Frontal decompression. Much solid growth removed. Uneventful recovery.

HISTOLOGY.—Fibrillary glioma.

RADIUM TREATMENT.—Seven days after operation 12 needles of 1 mgrm. were inserted into the brain at the periphery of the tumour and into the centre (*Fig.* 386). Radium left in position 7 days. Total, 2016 mgrm.-hrs.

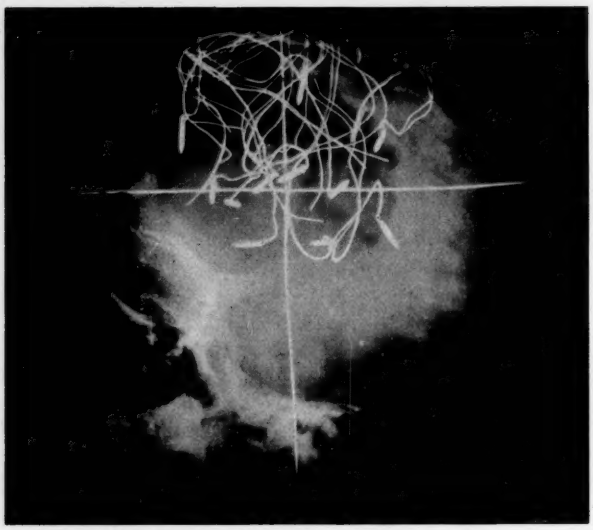

FIG. 386.—*Case* 10. Showing radium needles placed in and around tumour-bed; silver wires are attached to the needles.

AFTER-HISTORY (Nov. 14, 1930).—No fits; the operation area is concave; there is slight facial weakness on the left side; the discs are normal and vision is good. The boy goes to school, plays games, and has no disability.

TUMOURS OF THE CEREBELLUM.

Case 11.—C. R., male, age 12 years. (Dr. Gordon Holmes.)

HISTORY.—Admitted in May, 1930, with headaches, vomiting, diplopia, papill-œdema, and definite signs of a right-sided cerebellar tumour.

OPERATION. — Cerebellar decompression. Right cerebellar lobe found to be bulging. The cerebellum was incised and a small portion of the tumour removed.

RADIUM TREATMENT.—Eight gold seeds at 1·5 mc. each, screened by 0·5 mm. of gold, inserted (*Fig.* 387). The patient died seven hours after operation.

POST-MORTEM.—Examination revealed that the fourth ventricle was filled with gelatinous growth which extended upwards, dilating the iter of Sylvius and reaching downward to the foramen of Magendie.

HISTOLOGY.—The tumour is extremely reticular in structure in almost all parts, but towards the intact cerebellum, which is infiltrated rather than pushed aside by the tumour, it is more fibrous and frankly gliomatous in structure.

FIG. 387.—*Case* 11. Showing eight gold seeds in cerebellar tumour.

Case 12.—H. P., male, age 32. (Dr. Gordon Holmes.)

HISTORY. — The patient was admitted to hospital twenty months previously with nausea, headaches, staggering gait, diplopia, papill-œdema, and coarse lateral nystagmus; hypotonia of both arms; ataxy and dysdiadochokinesis on the left.

FIRST OPERATION.—Partial removal of left cerebellar tumour (Mr. Donald Armour). The patient was re-admitted with a bulging occipital tumour; increasing nausea and staggering and pain in fronto-temporal region.

HISTOLOGY.—Astrocytoma.

SECOND OPERATION (June 10, 1930).—The patient was again operated on by

Mr. Armour. The tumour was found filling the bone opening, part of it was cystic, and on the superficial wall of the cyst a number of rounded tumour masses were seen, varying in size from that of a pea to that of a walnut. The tumour was partly removed, two very large pieces being taken away.

RADIUM TREATMENT.—Ten needles of 1 mgrm. each were inserted peripherally around the site of the tumour, and 10 mgrm. into the centre, the threads being brought out of the wound. The radium was left in position for 7 days. Total, 3360 mgrm.-hrs.

HISTOLOGY.—Nature of tumour removed at the second operation: sarcoma rather than glioma.

AFTER-HISTORY (July 5, 1930).—Flaccid paraplegia. Absence of knee- and ankle-jerks. Difficulty in micturition. Two days later the paraplegia was complete, the abdominal reflexes were lost, and there was complete retention of urine. Evidence of tumour formation in the cord with steady deterioration of general condition.

Case 13.—A. S., male, age 14 years. (Dr. Gordon Holmes.)

HISTORY (March 31, 1930).—Three months' history of headaches and vomiting. Previously operated on for cerebral tumour in December, 1928; then history of headache, nausea, vomiting, and failure of vision for twelve months. Bilateral subtentorial decompression—cystic tumour opened in right cerebellar hemisphere. On discharge from hospital the neurological examination was eventually negative except for a questionable weakness of the left internal rectus. The patient remained well till three months previous to admission. He then began to vomit, at times incessantly; headaches and swelling on the back of the head (operation area). Discs pale. No retinal hæmorrhages. Motor system, no weakness. Co-ordination normal. Tends to fall to the right.

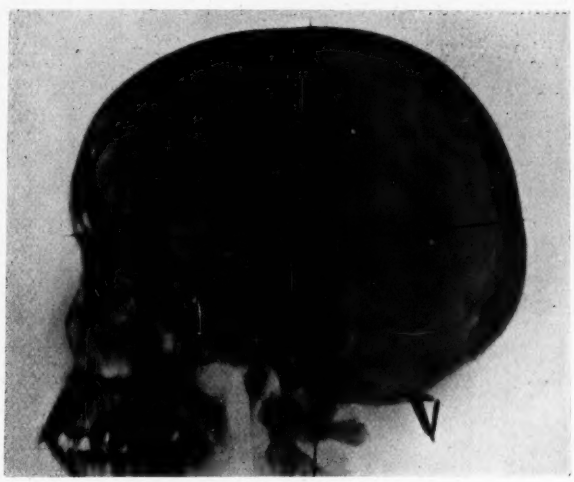

FIG. 388.—*Case* 13. Showing four needles in the cerebellar region.

OPERATION (May 9, 1930).—The old incision was opened and a cystic collection seen. On the medial wall of the cyst was a soft mass 4 by 5 cm., clearly tumour, in the substance of the destroyed right cerebellar lobe. The mass was removed.

RADIUM TREATMENT.—Four needles containing 5 mgrm. of radium element each were laid on the surface of the visible part of the tumour; the threads were led out of the wound (*Fig.* 388). Total dose in 7 days, 3360 mgrm.-hrs.

HISTOLOGY.—Fibrocellular glioma.

AFTER-HISTORY (November, 1930).—The patient is symptomless, vision is good, there is no motor weakness, and he has returned to school.

Case 14.—F. R., male, age 4½ years. (Dr. Birley.)

HISTORY.—Two months' history of severe headache and vomiting. Bilateral papillœdema.

OPERATION (July 2, 1930).—Cerebellar decompression, glioma in mid-line. Partial removal. Recovery and improvement.

HISTOLOGY.—Glioma with fairly well differentiated neuroglia fibres and large oval nucleus of the type of adult neuroglia cells. The cells are fairly closely placed. The neuroglial cells appear to be fully developed, and the tumour may therefore be classed as an astrocytoma fibrillare.

RADIUM TREATMENT.—Two months after operation. Columbia-paste cap made 15 mm. thick, 30 mgrm. of radium in 30 needles applied, 12 hours daily for 14 days. Total, 3600 mgrm.-hrs. Complete recovery of vision and motor function.

AFTER-HISTORY.—Uneventful recovery; remains well, and almost free from symptoms.

Case 15.—L. C., male, age 12 years. (Dr. Birley.)

HISTORY.—Two years' history of mental change, staggering gait, frontal headaches, deterioration of vision, occasional vomiting. Optic atrophy on right side; papillœdema left side. Nystagmus. Inco-ordination of both arms and legs.

OPERATION (June 26, 1924).—Tumour found in deeper portion of right frontal lobe. Partial removal.

RADIUM TREATMENT.—Insertion of 100 mgrm. of radium, left in position for 24 hours. Total, 2400 mgrm.-hrs.

AFTER-HISTORY.—Temporary improvement. Death from recurrence took place seven months later.

PITUITARY TUMOURS.

Case 16.—A. H., male, age 59 years. (Dr. Grainger Stewart.)

HISTORY.—Twelve months' history of failing vision and headache. Bitemporal hemianopia. Visual acuity: right, 6/12; left, cannot count fingers. Discs atrophic.

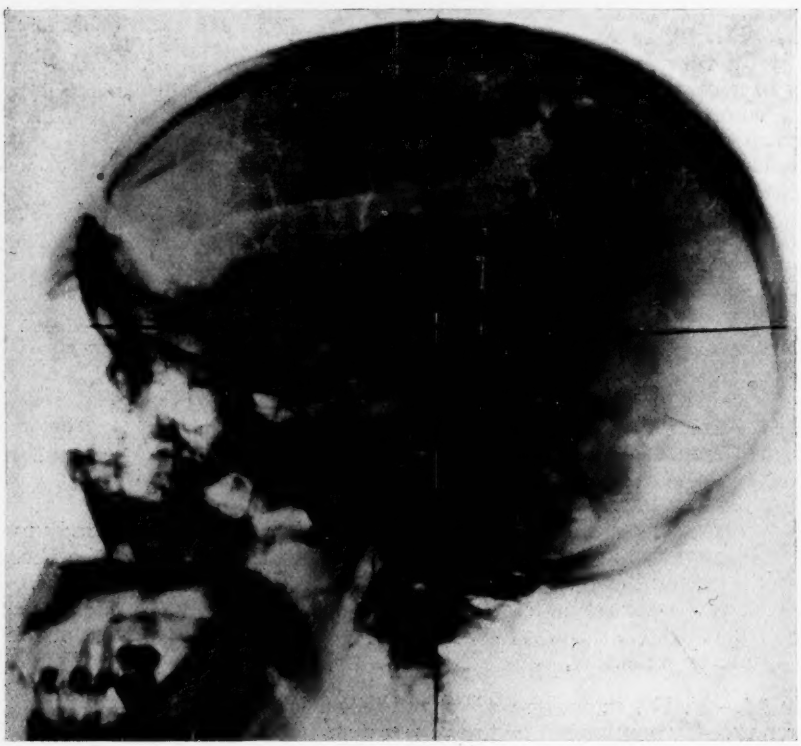

FIG. 389.—*Case* 16. Showing four gold seeds in the upper part of the pituitary fossa. The seeds were introduced through a frontal approach.

X RAY.—Nothing abnormal.

OPERATION (March 22, 1930).—Right-sided osteoplastic frontal flap. A supra-pituitary tumour, partly cystic, was exposed, and partially removed.

RADIUM TREATMENT.—Four radon seeds, 1·2 mc. each, screened by 0·5 mm. of gold, were inserted. Total, 633 mgrm.-hrs. (*Figs.* 389, 390).

HISTOLOGY.—Adamantinoma.

AFTER-HISTORY. — The patient remains well to date, but did not come up for examination.

FIG. 390.—*Case* 16. Antero-posterior view.

Case 17.—Dr. J. W., male, age 35 years. (Dr. Gordon Holmes.)

HISTORY.—Five months' history of failure of vision. Noticed that in reading the middle part of the word would be left out. Bitemporal hemianopia, and X-ray evidence of erosion of sella turcica.

OPERATION (July 30, 1930). —Right frontal osteoplastic approach. Large pituitary cyst found, containing brown fluid; this was punctured and evacuated by suction.

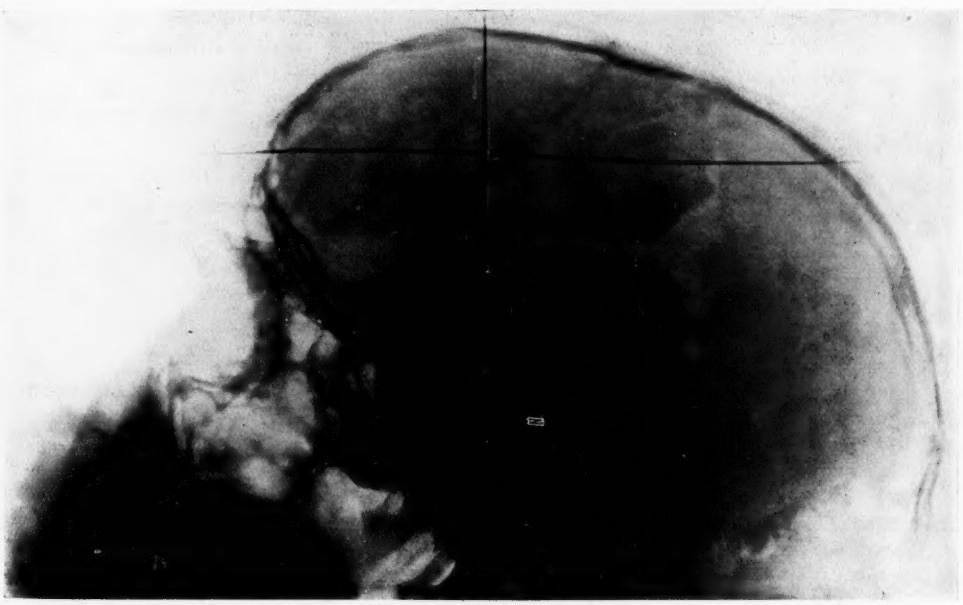

FIG. 391.—*Case* 17. Showing two radon seeds inserted vertically into the sella turcica through a frontal approach.

RADIUM TREATMENT.—Two radon seeds placed in the sella: 1·8 mc. each, screened by 0·5 mm. of gold (*Fig.* 391).

AFTER-HISTORY.—The patient is in good health and has returned to his practice with practically full vision.

Case 18.—G. N., male, age 58 years. (Dr. Kinnier Wilson.)

HISTORY.—Two and a half years' history of headache, diplopia, and polyuria. Bitemporal hemianopia; discs pale.

X RAY.—Skiagram shows enlargement of sella turcica.

OPERATION (April 24, 1930).—Frontal route: removal of a great deal of soft tumour.

RADIUM TREATMENT.—Insertion of 3 gold seeds containing 1·4 mc. each, screened by 0·5 mm. of gold.

HISTOLOGY.—Adenomatous hyperplasia.

AFTER-HISTORY (November, 1930).—General health good; very occasional diplopia; vision very greatly improved; no headaches, no polyuria.

FIG. 392.—*Case* 20. Showing two platinum seeds inserted by temporal decompression.

Case 19.—A. M., male, age 18 years. (Dr. Adie.)

HISTORY.—Three weeks' history of vomiting and headache. No fits, weakness of limbs, diplopia, or squint. Bitemporal hemianopia; blurring of upper margin of each disc.

X RAY.—Large pituitary fossa; probably suprasellar tumour.

OPERATION (April 25, 1930).—Right frontal osteoplastic flap: contents of a large suprapituitary partly cystic tumour evacuated.

RADIUM TREATMENT.—Four platinum seeds containing 1·5 mc. each, screened by 0·5 mm. of platinum, were inserted into the cavity.

AFTER-HISTORY.—The patient is in good health; has started work as a clerk; vision is very good; symptomless.

Case 20.—E. H., female, age 37 years. (Dr. Birley.)

HISTORY.—Three months' history of headaches, drowsiness, and failure of vision. Marked polyuria. Papillœdema, with +4D swelling and numerous hæmorrhages.

X RAY.—Shows destruction of posterior clinoid processes.

OPERATION (April 24, 1930).—Right temporal decompression. Cystic tumour observed in floor of third ventricle.

RADIUM TREATMENT.—Three radon seeds 1·8 mc. each, screened by 0·5 mm. of platinum, were inserted (*Fig.* 392). Good post-operative recovery.

AFTER-HISTORY.—The patient left hospital free from headache, with papillœdema subsiding, and vision improving.

Case 21.—D. A., female, age 30 years. (From the Surgical Unit, St. Thomas's Hospital.)

FIG. 393.—*Case* 21. Illustrating silver capsule introduced into the suprapituitary region.

FIG. 394.—*Case* 21. The same. Antero-posterior view.

HISTORY.—Five years' amenorrhœa, polyuria, and failure of vision. No papillœdema. Complete left temporal hemianopia. Enlargement of sella turcica.

OPERATION (Oct. 30, 1930).—Right frontal osteoplastic approach. Suprapituitary tumour exposed, incised, and much of its contents removed.

RADIUM TREATMENT.—One capsule containing 14 mc., screened by 1 mm. of silver, inserted (*Figs.* 393, 394).

HISTOLOGY.—Trabeculæ of tumour tissue are separated from one another by very numerous fibrovascular septa. In the intervening tumour tissue the cells have large irregularly-shaped nuclei. Kidney-shaped and sausage-shaped nuclei are frequently seen. Some nuclei stain faintly, others darker. The cytoplasmic matrix is abundant, indefinitely fibrous, and takes a yellowish colour with van Gieson's stain. In this and in the presence of numerous fibrils in a single process it does not quite resemble neuroglial tissue; nor do the characters of the nuclei suggest glioma. It appears to be a form of endothelioma.

AFTER-HISTORY.—Two months later the patient is practically well, with considerable visual improvement. It is a recent case, and is given here to illustrate the substitution of seeds by a capsule.

ANALYSIS OF RESULTS.

From the observation of the cases described and a large number of other cases treated, it seems clear that a certain percentage of patients appear to benefit from radium therapy. Although some cases are completely radio-resistant and do not respond to treatment, others respond rapidly both in general health and in functional recovery. The best results are obtained in astrocytoma and medulloblastoma. It is difficult to assess the value of radium treatment in oligodendrogliomata, as these tumours do well with surgical removal alone and the end-results are not yet available. Spongio-blastomata form the most interesting group. Clinically the most malignant, their response to irradiation is variable, some cases having proved successful, others complete failures; but in this group more than in any other post-operative surface irradiation appears indicated, as the survival-rate without radium is as a rule under one year, whilst with radium complete regression of symptoms has been obtained in a few cases.

CONCLUSIONS.

1. The effect of radium on normal brain tissue need not be feared, providing the dose given is within therapeutic range and adequate screenage is provided.

2. Interstitial irradiation has a very limited field of usefulness. The effect on the cerebral vascular system, both immediate and remote, must be kept in mind in planning the details of treatment.

3. Surface irradiation by the method described is at present the most useful form of treatment by radium and is of wide application. Maximum dosage over prolonged periods of time appears to be the optimum.

4. The effect of irradiation on the function of the choroid plexuses may prove of practical value, and deserves further study.

5. Decompression is essential for the various reasons stated.

6. Further study is to be directed to the relation between different types of tumour and their response to irradiation.

7. The desirability of repeating treatment even in the absence of symptoms at the end of a certain period of time may have to be considered. And the possibility of combining X-ray therapy with radium treatment offers a new field of investigation.

REFERENCES.

[1] Bailey and Cushing, *Tumours of the Glioma Group*, 1926.
[2] Greenfield, J. G., *Brain*, 1919, xlii, 1.
[3] Ross and Carmichael, *Proc. Roy. Soc. Med.* (Neurol. Sect.), 1930, May, 27.

SHORT NOTES OF RARE OR OBSCURE CASES

CYST OF THE ILEUM.

By R. Y. AITKEN,
SENIOR SURGEON TO BLACKBURN ROYAL INFIRMARY.

A boy, age 7, was admitted to the Blackburn Royal Infirmary on Feb. 5, 1930. He was brought to the Out-patient Department by his mother, who

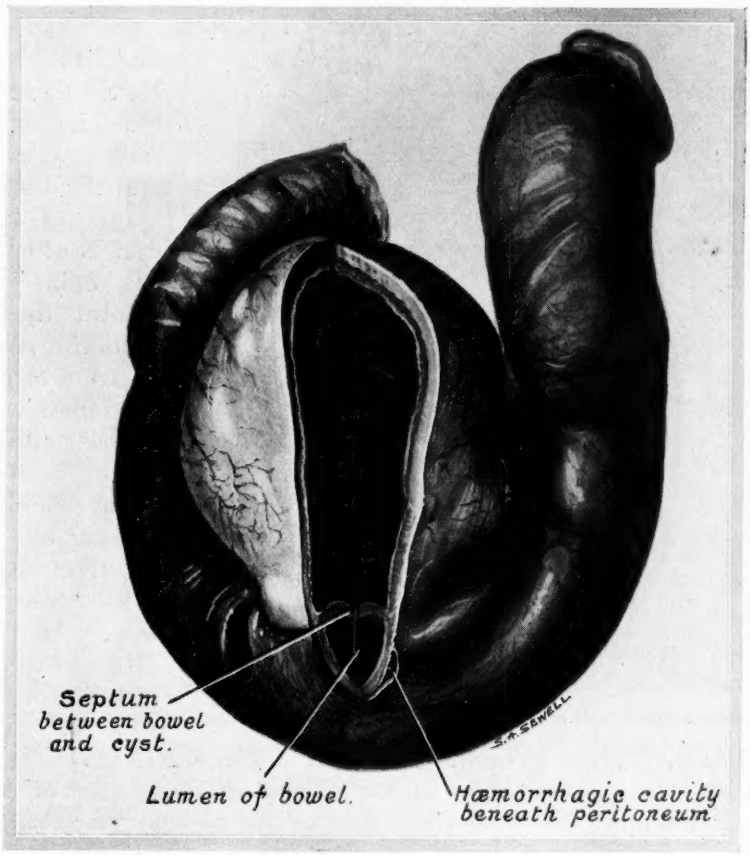

Fig. 395.—Cyst of ileum.

stated that he had been having attacks of abdominal pain and vomiting for the last six months. The medical attendant thought these attacks were due to mild appendicitis. The temperature was normal and the pulse 100.

On Examination.—The boy was found to be tender and slightly rigid over the right iliac fossa, and a mass could be felt low down in the fossa.

Operation.—The abdomen was opened through a right paramedian incision, and a loop of bowel of a deep purple colour, about nine inches long, was exposed, with a rounded cystic swelling 3½ in. by 2½ in. occupying the mesenteric border of the loop. The discoloration ended abruptly at either end of the affected gut. The loop and cyst were excised, and an end-to-end anastomosis established. The boy was very ill for several days as the result of acidosis, but ultimately made a good recovery.

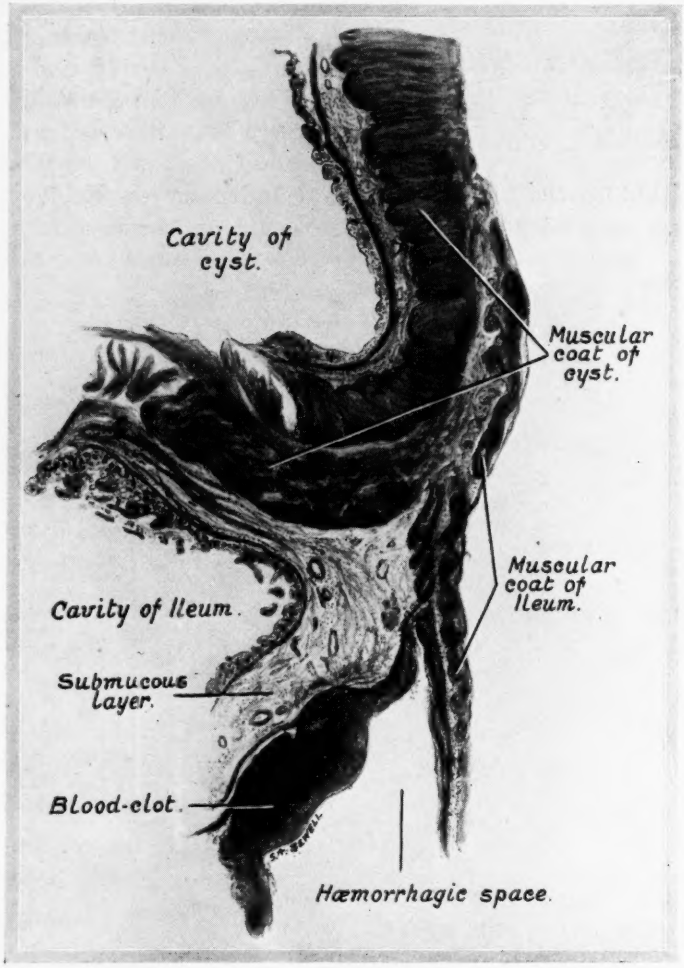

Fig. 396.—Section through the wall of the cyst and adjacent ileum.

The specimen was sent to Sir Arthur Keith, to whom I am much indebted for the report which follows, and for the drawings (*Figs.* 395, 396). The cyst is of embryological origin—a developmental diverticulum, with the cavity shut off from the lumen of the bowel.

Pathological Report.—A loop of ileum which was removed by operation together with an oval cyst measuring 3 in. by 1½ in. which is attached to its mesenteric border. At the site of attachment the bowel is markedly stenosed. The cyst has a thick muscular wall and is lined with mucous membrane with a villous surface in part of its extent; it has no communication with the lumen of the bowel. In seven inches of the length of the loop of ileum an extensive extravasation of blood has occurred into the wall of the bowel, separating it into an outer and inner layer. The purple discoloration of the peritoneum ceases abruptly near the extremities of the loop.

Microscopical examination of the cyst shows the presence of a thin mucous membrane with ill-developed villi and scanty, ill-developed glands;

the surface epithelium is for the most part desquamated. A well-marked muscularis mucosæ and submucous layer are present, and outside these is a double muscular layer of circular and longitudinal fibres. A section at the site of the union of the cyst with the bowel shows that the septum separating the two does not contain any muscular fibres of the ileum; the muscular coat of the latter can be traced from the bowel over the outer surface of the cyst. The original position of the cyst would thus appear to be in the submucous layer of the bowel.

RETROPERITONEAL CYSTS:

A REPORT OF A CASE OF A LARGE RETROPERITONEAL LYMPHANGIOMA.

By GEORGE D. F. McFADDEN,

ASSISTANT SURGEON TO THE ULSTER HOSPITAL FOR CHILDREN AND WOMEN, BELFAST.

Retroperitoneal cysts are of interest both from the clinical and pathological points of view. A cyst is rarely diagnosed, because it is rarely suspected. If the cyst becomes inflamed, the cause of the acute abdomen is as a rule only ascertained when the abdomen is opened. The anatomical relations of the cyst make it difficult to say from what structure it originates, and have led to error in its pathological classification; and the thinness of the cyst wall has not given the pathologist sound material on which to base his opinions.

On account of the absence of pathological proof, there has been much theorizing as to the origin of these cysts. Stoney[1] and Handfield-Jones[2] both mention as a possible origin the peritoneal cells that should normally disappear during the process of rotation and fixation of the colon. The anatomical relations of some cysts have given rise to the theory that they originate from cells in connection with the urogenital ridge. From its anatomical relations the case reported below might suggest such an origin; yet the piece of tissue taken on the second occasion showed, on microscopical examination, that the cyst was a lymphangioma. The tumour was very like that described as a lymphangioma by Gaudier and Gorse[3]; the clinical condition was similar to that in the case reported by Stoney in that the patient came under notice with the signs and symptoms of an acute abdomen. When Gaudier and Gorse described their case in 1913 they could find references to only four examples. Of late, however, these cases have aroused a good deal of interest and many excellent papers have been written describing them.[4, 5] Few observers have been able to identify definitely the origin of the cysts described, so for this reason the case reported below should prove of interest.

CASE REPORT.

History.—The patient was a boy of 5 years of age when first seen by me. He was brought to the Out-patient Department of the Ulster Hospital for Children, complaining of abdominal pain. The mother stated that he had

been operated upon for a right-sided hernia when 6 weeks old. (I afterwards found that it was a 'hydrocele' that had been removed.) After this operation the abdomen began to enlarge, though the enlargement caused no discomfort. Seven days before being seen by me the boy had an acute pain in the abdomen; at first it was more in the right side, but 'wore over' to the left. Relief was obtained if he got up on his knees and kept his head down on the bed. He vomited several times, was flushed, and sweated. The bowels were regular and there was no disturbance of micturition. The symptoms had been getting worse.

On Examination.—The boy was flushed; he lay with his legs drawn up, and had a greatly distended abdomen, the umbilicus protruding like a nipple. There was no venous engorgement or other abnormality of the abdominal wall. Both testes were in the scrotum, and there was no sign of a hydrocele or hernia. The abdomen felt tense, and the boy complained of pain on pressure over the lower abdomen, more marked over the right iliac fossa. No tumour could be felt, and the abdominal distension made detection difficult. The abdomen, except for the epigastrium and a small area below it, was dull on percussion. The temperature was normal, the pulse-rate 120. Nothing abnormal could be felt on rectal examination.

The mother stated that the size of the abdomen had not altered since the onset of the illness and had been the same for four years, causing no discomfort. A satisfactory diagnosis could not be made. All that could be concluded was that the child had developed some acute inflammation superimposed upon a chronic condition. Suggested diagnoses were an acutely inflamed appendix or an inflamed tuberculous gland in a case of tuberculous peritonitis. The symptomless distended abdomen existing for so long was unlike tubercle, and no satisfactory conclusion was arrived at. The child was admitted to hospital.

Operation.—Operation next day revealed a large inflamed retroperitoneal cyst that filled the whole of the lower abdomen. The cæcum, ascending colon, and small intestine were lifted up beneath the diaphragm. The cyst was horseshoe-shaped. In the middle line it reached about an inch above the bifurcation of the aorta; in the flanks it extended up to both kidney pouches. It reached down into the pelvis behind the mesosigmoid and the mesorectum, but did not touch the bottom of the rectovesical space. The peritoneum over the cyst was red and œdematous. The complete removal of the cyst was considered inadvisable, as it was difficult to strip, and the state of the child would not permit a lengthy operation. The peritoneum was therefore incised and stripped from the upper part of the cyst on the left side, and this part of the tumour was mobilized to bring it down to the anterior abdominal wall in the suprapubic region. The wall of the cyst so mobilized was fixed to the anterior abdominal wall and the redundant part cut off, leaving the cyst to discharge on the surface.

The cyst was multilocular; in one area colonies of small cysts projected outwards, giving the appearance of frog's spawn lying on the wall of the cyst. The fluid in the cyst was turbid, with flakes of lymph scattered through it. A piece of the wall examined under the microscope showed only fibrosis.

The boy made a good recovery from the operation. It was hoped that the inflammation might effect a cure.

Subsequent History.—About a year afterwards the child was admitted with the signs of acute intestinal obstruction. Operation revealed a band at the marsupialized area obstructing the small intestine. This was divided. An examination of the abdomen showed that there was still a moderate-sized retroperitoneal cyst present on the right side. The child made a rapid recovery and the parents were told to bring him back to hospital in three months' time for the removal of the remainder of the tumour.

Fig. 397.—A large irregular space is shown into which projects a nodule of lymphocytes showing a 'germ centre'. Plain muscle fibres are seen in the wall at one end. The fatty matrix contains also blood-vessels. (*Low power.*)

Three months later the growth was exposed. It was a bluish tense multilocular cyst, and extended from the lower pole of the right kidney and the third part of the duodenum down to the pelvis. A finger-like process extended into the inguinal canal. The cyst was closely adherent to the perinephric fat; the spermatic vein lay anterior to it. With care the whole cyst was completely removed. The child's convalescence was uneventful.

Fig. 398.—To show one narrow lymph-channel with incomplete lining of flat endothelial cells. Adjacent is a blood-vessel containing blood-corpuscles. (*High power.*)

The anatomical relations of the cyst as now seen suggested a close relation to the renal structures, and seemed to fit the necessary conditions for one arising from cells of the urogenital ridge. The second pathological report showed it to be otherwise. This report, for which I am indebted to Professor Drennan, shows the cyst to be definitely a lymphangioma.

Pathological Report (*Figs.* 397, 398).—The matrix consists mostly of fatty tissue, but in places it is fibrous with strands of plain muscle amongst the collagen fibrils. In this matrix is a sort of sponge-work

of channels, some long and narrow, some in the form of wide spaces. The lining of these is a single layer of flattened endothelial cells—absent in some parts—which are supported by fibrous tissue. Along many spaces are also plain muscle fibres from one to several layers thick. Collections of lymphocytes are seen, sometimes in the walls of spaces, sometimes lying irregularly in the meshes of the matrix, sometimes in the spaces themselves—more especially in the smaller channels. One nodule of lymphoid tissue, having a lymph-node arrangement, is seen in the wall of a space and only separated from the lumen by an endothelial layer. Blood-vessels, both arteries and veins, also occur running through the matrix, but are distinct from the spaces described above. There are scattered small foci of infiltration by polymorph leucocytes—evidently a superadded acute inflammatory reaction. The appearances are those of a cavernous lymphangioma, similar to the congenital 'hygroma' found in the neck and axilla.

Looking over the published papers and the descriptions of cases, this case would suggest that many retroperitoneal cysts are true lymphangiomas, and not cysts arising from local peritoneal cells or from cells derived from the urogenital ridge.

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[1] Stoney, R. A., *Brit. Jour. Surg.*, 1924–5, xii, 789.
[2] Handfield-Jones, R. M., *Ibid.*, 119.
[3] Gaudier, H., and Gorse, R., *Presse méd.*, 1913, xxi, 458.
[4] Hadley, H. M., *Surg. Gyn. and Obst.*, 1916, xxii, 174.
[5] Lloyd, E. I., and Higgins, T. T., *Brit. Jour. Surg.*, 1924–5, xii, 95.

CHRONIC SUBLUXATION OF THE DISTAL RADIO-ULNAR ARTICULATION.

By J. ALLAN BERRY, Napier, N.Z.

Chronic subluxation of the distal radio-ulnar articulation, or undue mobility of the ulna on the radius, is fortunately not a common condition following injuries about the wrist-joint, and when it does occur is generally so slight that there is very little inconvenience. There is, however, a class of case where it can result in a serious disability, the recognition of which in books dealing with fractures and dislocations is inadequate and the treatment described unsatisfactory. The operation described in the following case—namely, fusion of the inferior radio-ulnar joint, with resection of a portion of the lower quarter of the ulna to allow of pronation and supination—must have occurred to many surgeons, although I have not been able to find any account of it in the literature that is available to me.

C. A. Mc., age 20 years, was admitted to the Napier Hospital on July 17, 1929, suffering from a Colles's fracture of the right radius, associated with a separation of the styloid process of the ulna. Reduction of the fracture

was satisfactorily carried out, and a plaster splint was applied. Although the radius united, the fractured tip of the styloid process did not unite. The patient complained of the undue mobility in an antero-posterior direction of the ulna on the radius. Quite slight pressure on the palm of the hand would cause the head of the ulna to project backwards. As the patient was a carpenter by trade it was impossible for him to use a hammer, and in riding his motor-bicycle the vibration caused the bones to slip on one another, and this caused pain and swelling over the dorsal aspect of the inferior radio-ulnar joint.

Operation.—On Feb. 4, 1930, it was decided to fuse the inferior radio-ulnar joint and to resect a portion of the lower quarter of the ulna—the so-called Gallie's operation. This procedure was carried out by means of an incision over the ulna. The inferior radio-ulnar joint was exposed from the dorsal aspect, and its cartilage, which presented a fibrillated appearance, was removed. A bone-peg was then driven through the ulna into the radius. After this about an inch of the ulna in its lower quarter was removed.

Subsequent Progress.—The wound healed well and the patient in a short time was able to resume his occupation as a carpenter, being able not only to use a hammer without trouble, but also to mix concrete by means of a shovel, which he regarded as a more severe test of the stability of his wrist-joint. At first he complained of the slipping of the lower end of the upper portion of the ulna, but this uncomfortable feeling has now gone. Supination and pronation of the hand are perfect; in fact, the range is rather greater than on the other side. He plays hockey without noticing any disability.

This type of operation would probably be of value in a number of other conditions. In those cases of marked shortening of the radius due to an old Colles's fracture, with great prominence of the lower end of the ulna, leading to pain on rotation of the forearm, it seems likely that the result following an operation such as that described would be better than removal of the prominent end of the ulna. In certain cases of crossed union it would probably be of distinct value in restoring pronation and supination; also in Madelung's deformity the operation should prove useful.

Carson's *Operative Surgery* (p. 87) makes the following statements when dealing with the inferior radio-ulnar joint. "Arthroplasty of this joint has been replaced by Gallie's operation. It is a matter of common experience that fractures of the lower quarter of the ulna have a strong tendency to non-union. Profiting by this, Gallie advised treating ankylosis of this joint by the simple procedure of excising an inch of the lower quarter of the ulnar shaft with the periosteum."

A similar statement is made in the text-book of Rose and Carless. Professor W. E. Gallie in a personal communication states that the operation of removing a segment from the ulna near its lower extremity is not his, and he has not been able to discover how the operation got into the literature under his name, as he has never used the procedure or written about it.

TWO UNUSUAL CASES OF STRANGULATED HERNIA.

By ANDREW FOWLER,

ASSISTANT SURGEON TO THE ROYAL INFIRMARY, ABERDEEN.

Case 1.—The following case of reduction of hernia 'en masse' appears to me to be of sufficient interest to warrant its being placed on record.

History.—The patient, a male, age 53, was admitted to the Aberdeen Royal Infirmary. He complained of a severe degree of pain in the lower abdomen that had lasted fourteen hours. He had suffered from a 'rupture' on the right side for as long as he could remember. This had been continually increasing in size, but he could always push it back without difficulty; a truss had never been worn. While loading a cart with sheaves about 7 o'clock on the morning of admission the patient felt something suddenly give way in this region. The swelling grew much bigger and could not be pushed back. He persisted in his efforts, however, to reduce the swelling by manual pressure, and after about an hour, to use his own words, made it disappear with great difficulty and much pain. He felt better afterwards and had some food, but a little later felt sick and vomited. He was seen by his doctor, who advised hot applications to the abdomen. He vomited twice later in the day, and his doctor on again seeing him in the evening sent him to hospital, having diagnosed strangulated hernia with 'reduction en masse'.

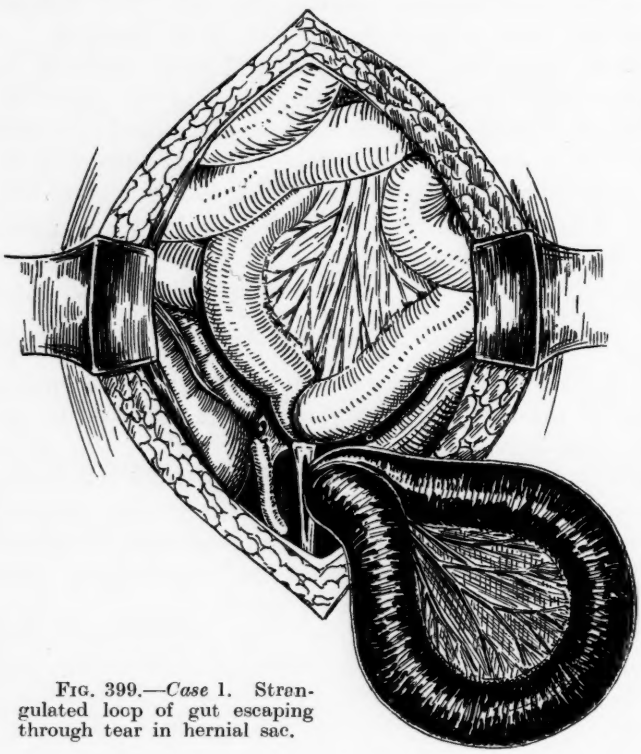

Fig. 399.—*Case* 1. Strangulated loop of gut escaping through tear in hernial sac.

On Admission.—The patient did not look seriously ill, but was rather cold after an eighteen-mile journey. His pulse was 64, respirations 22, and temperature 97·4°.

On Examination.—His tongue was slightly coated all over with a yellowish fur. The abdomen moved fairly well on respiration, but on the right half the movement was definitely less than on the left. No inguinal hernia was visible, but on coughing a small rounded bulging appeared over the

situation of the right internal abdominal ring. On passing a finger along the inguinal canal a firm cord could be felt running obliquely upwards from the outer side of the internal ring, and was lost in the abdominal cavity. On palpating the abdomen a definite area of tenderness was made out in the right lower quadrant, with its maximum intensity slightly to the left of McBurney's point. There was also some tenderness in the epigastrium. Muscular rigidity was also well marked in the right lower quadrant, but in spite of this, a well-defined rounded mass about the size of an orange could be made out, occupying the right iliac fossa. On rectal examination nothing abnormal was made out.

OPERATION.—Operation was decided upon, and under open ether anæsthesia the abdomen was opened by a low right paracentral incision, retracting the rectus muscle outwards. On opening the peritoneum a quantity of blood-stained fluid escaped, and after packing off the small intestine and cæcum a black mass was seen in the right upper pelvis. This proved to be a loop of small intestine lying free in the peritoneal cavity. On searching for the site of strangulation this was found to be the neck of the hernial sac which had been forced back into the abdomen. The strangulated loop had escaped through a tear extending through the wall of the sac and parietal peritoneum, as shown in the diagrammatic sketch (*Fig.* 399).

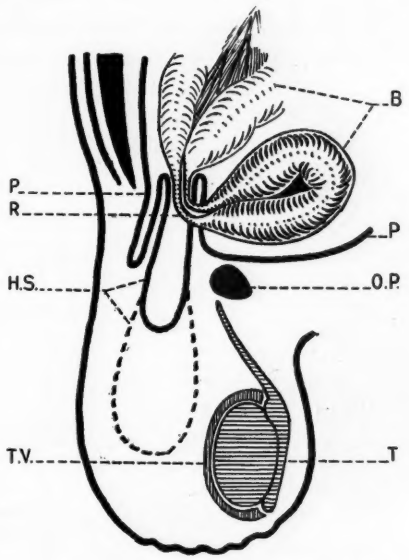

FIG. 400.—*Case* 1. Condition found at operation. P, Parietal peritoneum; R, Rupture through neck of sac; HS, Hernial sac; TV, Tunica vaginalis; T, Testis; OP, Pubis; B, Bowel.

The constricting band was divided, and on examining the sac the double tear of sac and parietal peritoneum was confirmed. The loop of small intestine was wrapped in saline gauze and left for ten minutes, during which time the sac was excised; the internal abdominal ring and the tear in parietal peritoneum were closed with a double purse-string suture of linen thread. The loop of bowel, about ten inches in length, did not recover sufficiently to justify leaving it, and was therefore resected. End-to-end anastomosis was performed about four feet above the ileocæcal junction. The abdomen was closed in the usual manner without drainage, and the patient made an uninterrupted recovery; he was discharged on the seventeenth day.

The difficulty in making an accurate diagnosis in this case was due to the fact that one could hardly credit the man's statement of having reduced the hernia while there was still the appearance of a bubonocele in the right inguinal canal, and, without the history, the diagnosis of acute appendicitis with abscess might readily have been made on the physical signs. Several accidents of over-zealous taxis are described in recent surgical text-books,

and a specially clear description with diagrams is given in Beattie and Choyce.[1] The conditions found in this case do not seem to correspond to any of the types described therein. I have attempted to reproduce diagrammatically after the manner of the illustrations in the above-mentioned textbook the condition actually found in this case (*Fig.* 400).

Case 2.—The second case is interesting from the history and subsequent findings at operation.

History.—H. U., female, age 66, was admitted to Aberdeen Royal Infirmary suffering from right-sided abdominal pain and a painful swelling in the right groin. The abdominal pain commenced three days before admission. At first it was generalized—of a dull aching character—accompanied by nausea, but without vomiting. This pain remained during the first day with no disturbance of micturition, and the bowels moved normally. The following day the pain gradually localized itself to the right iliac fossa, increasing in intensity until it became very acute, with occasional bilious vomiting. The bowels moved on this day following an aperient, with no unusual character of the motion. There was no radiation of the pain and no disturbance of urinary function. On the morning of admission the patient complained of a small painful swelling in the right groin which she had not previously observed. A slow increase in the size of the swelling occurred, and the pain in the right iliac fossa continued, but seemed to have some relation to the painful swelling in the groin noted above. On this day the bowels again moved normally. She was admitted with a diagnosis of strangulated femoral hernia.

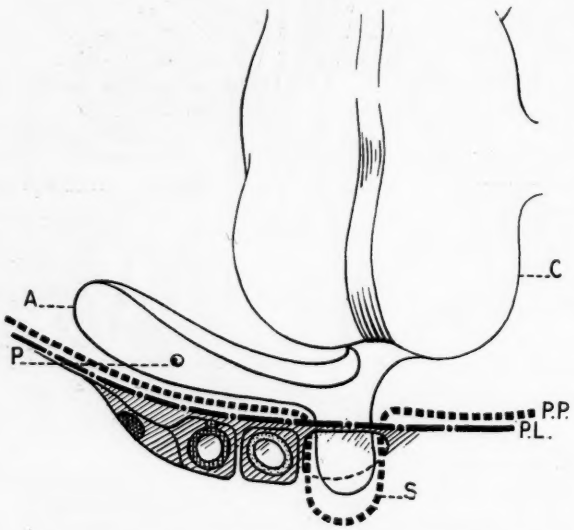

Fig. 401.—*Case* 2. Condition found at operation. A, Appendix; P, Perforation; S, Sac; PL, Poupart's ligament; PP, Parietal peritoneum; C, Cæcum.

On Examination.—The pulse-rate was 100 per minute, and the temperature 99°. The patient appeared healthy and well-nourished; the tongue was very furred and the breath foul. The abdomen was symmetrical, and moved fairly well on respiration except in the right lower quadrant. Moderate pressure in the right iliac fossa elicited marked tenderness, which extended upwards to the umbilicus. Marked rigidity of both abdominal recti in the lower halves was apparent. Dullness on percussion was made out in the right flank. There was nothing further of importance to note in the abdominal examination. A rounded swelling about an inch in diameter was observed at the site of a right femoral hernia. This swelling was extremely tender;

it could not be reduced, it was dull on percussion, and there was no impulse on coughing.

OPERATION.—Operation was decided upon immediately, and under open ether anæsthesia a three-inch incision was made over the swelling parallel to Poupart's ligament. A small femoral hernial sac was found after removal of much fat. On opening the sac a tiny piece of what appeared to be gangrenous intestinal wall was visible. This was held gently with a forceps, and the neck of the sac divided with Gimbernat's ligament. The gangrenous portion of intestine on being pulled down was found to be the appendix, the distal two-thirds of which was lying free in the abdominal cavity in a gangrenous and perforated condition. The appendix was removed in the ordinary way by slightly enlarging the neck of the sac, and a fair amount of foul-smelling pus escaped from the femoral hernia opening. The right iliac fossa was drained through a stab incision just inside the anterior superior iliac spine and through the hernial opening. The patient made an uninterrupted recovery. The operation wound healed firmly by granulation, and further operative repair of the crural canal is contemplated at a later date should it prove necessary. From the accompanying diagrammatic sketch (*Fig.* 401) the condition at operation is seen, and the coloured drawing (*Fig.* 402) shows the appendix after removal.

FIG. 402.—*Case* 2. Appearance of appendix after removal.

The interesting features of this case are: (1) The fairly obvious history of an attack of acute appendicitis with the appearance of a very recent strangulated femoral hernia, giving rise to difficulty in deciding which condition was responsible for the majority of the symptoms; (2) The absence of symptoms of intestinal obstruction; (3) The finding of a condition in the appendix very similar to a Richter's hernia of the intestine; (4) That the partial strangulation of the appendix in the manner described above was responsible for the appendicular symptoms.

I am greatly indebted to Dr. F. J. Bowie and Mr. R. Ogg for having prepared the drawings, and to Mr. G. H. Colt for permission to publish the cases.

REFERENCE.

[1] BEATTIE and CHOYCE, *System of Surgery*, ii, 657.

REVIEWS AND NOTICES OF BOOKS.

Sensation and the Sensory Pathway. By JOHN S. B. STOPFORD, M.D., F.R.S., Professor of Anatomy, University of Manchester. Demy 8vo. Pp. 148 + xii. Illustrated. 1930. London: Longmans, Green & Co. Ltd. 7s. 6d. net.

PROFESSOR STOPFORD has made a most readable summary of the present state of knowledge of the sensory pathways, with particular reference to the two-stage recovery of sensation after suture of peripheral nerves. The process of regeneration of sensation is carefully described, including the author's own contributions on the recovery of deep sensation. The sensory nervous pathway in spinal cord and brain-stem, and the attributes of thalamic and cortical sensation are outlined.

As so much controversy has centred round the interpretation of the two-stage recovery of sensation, Professor Stopford's view of this is interesting and stimulating. He thinks that the disturbance of intraneural pattern inevitable in regeneration is responsible for the sensory dissociation, and his studies of cases of recovery after nerve compression (without actual severance), and the studies of Sir Edward Schafer on recovery after compression, strongly support this view. Professor Stopford believes that end-organs are specific in function, and that the sensory nerve-fibres (by virtue of their connections) are also specific in function. To explain the phenomenon of reference of sensation occurring in the first stage of recovery, he extends this specialization to the nerve-fibres conveying the elements of second-stage (epicritic) sensation—a procedure which necessitates the postulation of a fibre to appreciate localization ('localization' fibre). To the reviewer it would seem that this postulation of pathways for abstractions of sensation is unnecessary, and that it would be more physiologically sound to regard the whole of second-stage sensation simply as the possibilities for integration which precision in localization gives. The well-authenticated multiple fission of regenerating nerve-fibres might well be used to explain reference and hypersensitiveness, on the basis of more widespread innervation by any one fibre and excess of innervation at any one point. If regeneration of nerve-fibres in heterogeneous channels is responsible for the two stages of recovery of function, it seems that the impossibility of anything approaching accurate microscopical apposition of the ends of nerve-fibres of specific function precludes the attainment of complete functional regeneration.

Congenital Club-Foot (Talipes Equinovarus). By E. P. BROCKMAN, M.Chir., F.R.C.S., Orthopædic Surgeon, Westminster Hospital, etc. Demy 8vo. Pp. 110 + viii, with 92 illustrations. 1930. Bristol: John Wright & Sons Ltd. 10s. 6d. net.

THIS book is in many respects a model of what a monograph should be. It gives the best account of congenital club-foot that has been published since Walsham and Hughes's *Deformities of the Human Foot*, which appeared in 1898.

The historical survey of the literature divides it into three periods: the first, from ancient times down to the introduction of tenotomy into England by Dr. Little; the second, from that time to the publication of Walsham and Hughes's book, which, as the author points out, is disappointing in that it deals so largely with the minute changes found in the bones of specimens of an advanced or an untreated stage of the deformity; and the third period, from 1900 down to the present time.

With regard to the pathological anatomy, the author's conclusion is that the deformity is due to a displacement of the anterior portion of the os calcis downwards

and inwards beneath the head of the astragalus, and displacement of the scaphoid downwards and inwards until it approximates to, or even touches, the sustentaculum tali or the internal malleolus. By these displacements the head of the astragalus is left very largely uncovered, and only articulates with a limited portion of the socket into which it should enter. The alterations in position of the scaphoid and os calcis with regard to the astragalus are only those which occur in the normal foot when it is adducted, inverted, and plantar flexed, but they are exaggerated in degree. This leads to the view that the principal structural defect is a congenital subluxation of the head of the astragalus within its acetabulum, with shortening of the muscles which control this socket. Emphasis is therefore placed upon the joint displacement and contraction of muscles and ligaments, in opposition to the view that the deformity is primarily bony, which was taken by Walsham.

With regard to the etiology, after a survey of the possibilities of (1) Defect in the germ, (2) Arrest of development, (3) Spasmodic muscular action, and (4) Mechanical causes in utero, the conclusion is that congenital club-foot is caused by a primary failure in development to their full extent of all the tissues of the foot, and that we know nothing of the influence that controls development, or why it should sometimes fail partially or completely.

The methods of treatment described in detail are naturally those which the author himself favours. He is inclined to the use of adhesive strapping as a method of correction in the infant—a method which can undoubtedly have admirable results in the hands of someone who is prepared to devote the time and attention required. The difficulty of this method is that the manipulation and strapping must be repeated at frequent intervals—three times a week is given as ordinarily sufficient in the early stages. The procedure is therefore difficult to carry out except with children who are in hospital or who can be brought up with great regularity; and in private practice it is only possible when a trained orthopædic nurse can be supplied or when the parents can afford very frequent attendances at the orthopædic surgeon's rooms. Manipulation with fixation in plaster-of-Paris is advocated for children whose treatment begins when they are a few months old, but little detail is given as to the exact method and position of fixation. For older untreated or relapsed cases the author describes the method of open correction by division of the structures on the inner side which bind together the scaphoid, the sustentaculum tali, and the internal malleolus until the scaphoid can be abducted and replaced in its proper position in relation to the head of the astragalus. He naturally condemns bone operations, which should only be carried out as a last resort. For choice, if a bone operation is required, he advises an arthrodesis of the tarsal joints, with removal of as much bone as is necessary to correct the deformity.

Chirurgie des Kindersalters. By Prof. Dr. R. Drachter, Leiter der chirurgischen Abteilung der Universitätskinderklinik, München; and Dr. J. R. Gossman, Assistenzarzt der Abteilung. Third edition, fully revised and enlarged. Crown 4to. Pp. 1031 + xvi, with 714 illustrations. 1930. Leipzig: F. C. W. Vogel. Paper covers, M. 125; bound in half leather, M. 135.

This text-book of the surgery of childhood is an excellent contribution to a highly important subject. The work is fully inclusive—no matter of importance has been omitted, and in many instances the wealth of detail is almost embarrassing. Professor Drachter contributes the general and introductory matter, and included in this chapter are the recommendations regarding anæsthesia. Close attention is paid to avertin narcosis, and, while views expressed are non-committal, this method has evidently met with favour; in fact it would seem that the dangers of an unduly low blood-pressure are less evident in the child than in the adult.

The section dealing with errors of the mouth, face, and jaw is beautifully and profusely illustrated. The operative methods recommended are those in general use. In cleft palate the Langenbeck-Ferguson procedure is advised, and an appropriate warning is given regarding the risks of forcible alveolar or palate moulding by means of plates or wires. It is regrettable that in this otherwise excellent book

a method of retrograde œsophagoscopy and œsophageal dilatation should be described and figured which is obviously unsuitable to clinical practice and experience. The authors would do well to reconsider this section.

The chapters dealing with abdominal conditions are less detailed than one would have anticipated. Such an important subject as congenital volvulus and gut-rotation errors might have received a fuller consideration; the section on congenital stenosis of the pylorus is also disappointing. Hernia is fully discussed and well illustrated, but the method of applying a dressing to the wound of an inguinal hernia is not so efficient as that employed in this country. The account of the treatment of empyema is critical and satisfying. Rib resections are not favoured, but the value of cannula puncture and suction drainage is stressed. It would have been appropriate to describe the method of 'closed drainage', and fuller recognition might have been given to the work of Graham in relation to the dangers of mediastinal mobilization.

The orthopædic sections of the book are uniformly good with the exception of the account of the pathology and clinical features of acute osteomyelitis. Insufficient attention has been paid to the recent work on the origin and development of the disease.

One of the peculiar values of the book is the inclusion at the end of each chapter of a recent bibliography. It is a plan which enhances the value of a publication, and it might well be adopted more widely.

To sum up—the volume is an excellent presentation, and if some sections are open to criticism, it is because they stand out in contrast to the excellence of the general whole.

Some Aspects of the Cancer Problem. An account of researches into the nature and control of malignant disease commenced in the University of Liverpool in 1905, and continued by the Liverpool Medical Research Organization (formerly the Liverpool Cancer Committee), together with some of the scientific papers that have been published. Edited by W. Blair Bell, B.S., M.D. (Lond.), F.R.C.S., Hon. F.A.C.S., Fellow of King's College, London; Director of the Liverpool Medical Research Organization, etc. Imperial 8vo. Pp. 543 + xiv, with 90 plates. 1930. London: Baillière, Tindall & Cox. 63s. net.

The object in producing this large volume has been to render accessible all published information on the lead treatment of malignant disease and other connected problems. The method adopted has been to reproduce papers published by the members of the Liverpool Medical Research Organization, with minor alterations, and some amplification of recent case-histories, these papers being classified and brought into sequence, while the whole production is cemented by an introduction, short interpolations, and a general summary. The book is, in fact, a reprint on an almost unprecedented scale, and for those who have followed the history of lead treatment in publications and discussions of the last eight years it contains very little that is new. This method of presentation, although admittedly it serves the subsidiary purpose of illustrating the evolution of its subject, is an unsatisfactory one for the reader whose object is to grasp well-ascertained and up-to-date conclusions. The book gives an even more fragmentary impression than might be supposed, owing to the inclusion not only of short papers of lesser importance, but of short excerpts from discussions, many of which are of a purely speculative character. Repetition is necessarily frequent. To have re-written the whole of this work would have been a formidable task, but the condensation so effected, and perhaps the results of the critical review of the whole subject which re-writing would enforce, could certainly have improved its form and enhanced its value.

The need for reconsideration is most evident in the chapter dealing with the actual treatment of malignant disease in the human subject, which is made up chiefly of two articles previously published by Professor Blair Bell, the second being here greatly amplified, and including the histories of about seventy successful cases. The actual number given in a subsequent table as 'believed cured' is 51. Although 566 cases have now been treated, there is no attempt at an analysis of the factors

controlling success or failure. It should, for example, be possible to deduce a relationship between the results of treatment and the type of growth treated. There are theoretical grounds for supposing that the more malignant a tumour is, the more susceptible it will be to the action of lead, and a remarkable number of the successful cases described appear to have been either sarcomas, or carcinomas in comparatively young subjects. This aspect of the question is not even discussed, and no attempt is made to classify results according to the type of growth, or in relation to any other factor which might affect success. Nor are figures given to indicate what proportion of the successful results followed treatment by lead alone and lead combined with X rays : it is surprising that steps should not have been taken to disarm the frequent criticism which has centred on this point. In the series of case-histories there are five categories, one of which is made up of cases treated by lead and X rays combined, but there is no statement that these were the only cases so treated, and at least one other case appearing in a different category is stated to have had X-ray treatment. There is no adequate description of the technique of treatment : the only precise references to dosage and intervals (p. 404) are in an article published in 1926, when experimentation " with different quantities and intervals " was still in progress : the results of this do not appear to be given anywhere later in the book.

These omissions serve to emphasize the unsatisfactory results to be expected from compiling already published papers of different dates, and it is to be hoped that in future editions a critical review of the results achieved may be inserted, with an analysis of results in relation to type and extent of growth, age, quantity of lead given, and nature of any associated treatment. Only by such data can the experience gained in Liverpool be made available elsewhere.

The mode of action of lead on the malignant cell appears still to be imperfectly understood. That a form of coagulation necrosis may be produced has been demonstrated repeatedly (though not consistently), but a detailed histological study at different stages of the process, such as has been made to elucidate the action of radium, still remains to be carried out. The relative concentration of lead in the growth and other tissues forms another question which apparently awaits an answer. The number of analyses of growths and other tissues made is too small, and the results are too variable, to permit of the conclusion that malignant tissue has a special affinity for lead. These are the experimental lines on which progress might well be achieved. When the action of lead is more clearly understood, and when it becomes possible to identify the type of case which will respond to it, the striking success achieved in some of the cases so far treated may well become more common.

The book is cumbersome, but well produced and profusely illustrated. The proceeds of its sale are to be paid into the funds of the Liverpool Medical Research Organization.

Radiologie clinique du Tube digestif. Published under the direction of PIERRE DUVAL, J.-CH. ROUX, and H. BÉCLÈRE. II. Œsophagus, Intestine, Liver, etc., by J. GATELLIER, Professeur agrégé à la Faculté de Médecine de Paris ; F. MOULIN, Chef de Laboratoire à la Faculté de Médicine de Paris ; P. PORCHER, Chef de Laboratoire de Radiologie à la Faculté de Médecine de Paris. Fasc. 1 and 2. Royal 4to. Pp. 389, with 823 illustrations. 1930. Paris : Masson et Cie. Bound in two volumes for consignment abroad, Fr. 330.

THESE superb volumes form the second portion of a complete work upon the clinical radiology of the digestive tract. The first section on the stomach and duodenum appeared in 1928, and a large edition was rapidly exhausted. We have no doubt that the present volumes have a similar fate in store. The volumes consist of a series of 416 radiographs showing all portions of the intestinal tract under both normal and pathological conditions. The plates are of the highest order and have been selected with great care, and they are so numerous that it has been possible to show every pathological condition under numerous aspects. Facing each photographic plate is a diagram which shows in outline the particular features for which the plate has been selected, whilst beside the diagram is a brief explanation of what it represents. The diagrams are drawn with very great skill, and represent with

absolute faithfulness the characteristics of the photographs, so that the two together furnish a pictorial clinical pathology of the intestinal tract.

This in itself would be a very considerable achievement, but it is supplemented by a running commentary in which the broad clinical pathology underlying the various conditions is described with a brevity and precision characteristic of the best French scientific literature. Each group of photographs is introduced by a precise description of the technique required in the examination of this particular region. The preparation of the patient is described in accurate detail, and various technical difficulties which may arise are discussed. The various methods devised for rendering this portion of the intestinal tract visible under the X rays are described and criticized, and here the practical details which are introduced are often of incalculable value and will make all the difference between success and failure to those who attempt to follow the procedures advised.

Finally, the most careful details are given of radiographic technique, so that even the most inexperienced can scarcely go astray. It is a pleasure to congratulate our French colleagues on such a brilliant production, as useful to the beginner as to the expert. The former could not make a better investment, for these volumes will save him from many pitfalls; and we venture to think that there is no one so expert but that he will find much to arouse his interest and to assist him in his work. The enthusiast will treasure them as a supreme example of his art and of the ingenuity which can be exercised in its application.

Surgical Diseases of the Thyroid Gland. By E. M. EBERTS, M.D., Surgeon to the Montreal General Hospital; Associate Professor of Surgery, McGill University. With the assistance of R. R. FITZGERALD, M.D., and PHILIP G. SILVER, M.D. Crown 8vo. Pp. 238 + xii, with 48 illustrations. 1929. Philadelphia: Lea & Febiger. $3.50 net.

THE authors state in the preface that the aim has been to present the subject in a practical way, and within reasonable compass. It may be said at once that they have succeeded admirably. On the clinical side the views are based upon work carried out in recent years in the Goitre Clinic of the Montreal General Hospital by Eberts and his associates. The co-ordination of the work of these associates and assistants has been mutually helpful, and it is obvious how careful the study of the material has been. The terminology of the goitrous thyroid gland is far from being standardized—a survey of the most recent literature will show how diverse personal views still are—so that the observations of a man with the prestige of Eberts, working with capable assistants in a large teaching centre, merit attention. The extent of the material contained within 238 pages is very great. The author has a rare faculty for compression combined with lucidity. The chapters on embryology, anatomy, histology, physiology, and pathological physiology are excellent. The gross and microscopic pathology of the thyroid gland present difficulties to all writers. The author has followed the classification suggested by Williamson and Pearse, in so far as it refers to trophic and plastic lesions, but he does not use this as the basis of his clinical classification. Since some types of thyroid disease are peculiar to certain countries, a uniform classification is naturally difficult. That given is the one which has appeared most suitable for the types of cases met with in the province of Quebec, and Eberts believes it to be generally applicable to the continent of America. The etiology and prophylaxis of simple goitre are well discussed.

The chapters on the subject of Graves' disease are good. The pre-operative and post-operative management given is sound, the risks of operation are clearly set forth, and the case-reports selected to illustrate important points. A chapter is given to the differential diagnosis between Graves' disease and toxic adenoma. A good many writers in different countries believe that the differences are not essential, and are dependent on the extent to which non-toxic pathological change has preceded the onset of the toxic condition. The chapters given to thyroiditis and malignant disease are adequate. The statement that " were examinations less

perfunctory more cases of malignancy would be recognized or suspected before glandular metastases had occurred " is timely, and the necessity for very careful examination of every adenomatous nodule removed is emphasized. Altogether this is a good book.

Lehrbuch der Chirurgie. Dedicated to Professor VON EISELSBERG by his pupils. Edited by Professors P. CLAIRMONT (Zürich); W. DENK (Graz); H. VON HABERER (Düsseldorf); and E. RANZI (Innsbruck). Royal 8vo. In two volumes. Vol. I. Pp. 869 + xiv, with 389 illustrations. Vol. II. Pp. 658 + xiv, with 298 illustrations. 1930. Vienna: Julius Springer. Paper covers, RM. 66; bound, RM. 69.80.

IT must be a proud and happy day for any teacher when he can have such a work as this written, presented, and dedicated to him by his own pupils. It is a comprehensive text-book covering the whole field of general surgery, including a short account of typical operations, but not dealing with the specialities of gynæcology or ophthalmology. The book is on the ordinary lines of an advanced student's text-book, and is well illustrated by line drawings and very clear diagrams. The opening chapter on general surgery is short and concise, covering in forty-four pages the subjects of wounds, inflammation, asepsis, anæsthetics, and the conduct of an operation. There is no special chapter dealing with specific infections—tubercle or syphilis, for example—although of course these diseases are referred to under the affections of special organs.

Roentgenology is dealt with at some length, with an adequate account of deep X-ray therapy. The different organs with their injuries and diseases are described systematically—namely: the blood-vessels, the skull, the central and peripheral nervous system, the face, the jaws, the neck, the nose and larynx, the ear, the mouth and salivary glands, the thorax, the breast, the abdomen, stomach, intestine, appendix, rectum and anus, peritonitis and ileus, the biliary apparatus, liver, pancreas, spleen, hernia, urology (including the male genital organs), the vertebræ, the pelvis, soft parts of the limbs, bones and joints, including fractures and dislocations.

It is hardly possible to appraise or to criticize in any detail a work of this size, but we welcome it as being an authoritative and complete German text-book compiled by leading surgeons who have all received their training in the famous school of Vienna.

Physikalisch-Chemische Probleme in der Chirurgie. By Dr. C. HÄBLER, Privatdozent für Chirurgie in Würzburg. 9″ × 6½″. Pp. 274, with 62 illustrations. 1930. Berlin: Julius Springer. RM. 19.60.

THE contents of this work may be divided into three parts: (1) The physical chemistry of the body; (2) The application of this to 'general' surgery; and (3) The application to the surgery of special organs. The amount of ground which the author has attempted to cover is enormous, and probably could not be dealt with really satisfactorily in a volume of this size. In consequence, most of the first part will be found very difficult reading by anyone without a fairly extensive previous knowledge of the physical chemistry of the body. The research worker in surgery or surgical pathology will find much material for thought, though he may not regard some of the problems dealt with—for example, that of œdema—as quite so simple as would appear from this book.

The approach to some subjects is different from that usual in this country. For example, in assessing renal efficiency in connection with operability the author carries out as a routine the determination of the freezing-point depression of the blood, the estimation of the 'rest-N', and a refracto-metric estimation of the serum protein.

This book would be useless to the surgeon who had not a proficient knowledge of chemistry.

Lehrbuch der Mund- und Kieferchirurgie. By Prof. Dr. ERICH SONNTAG, A.O. Planmässiger Prof. der Chirurgie und Direktor des Chirurgisch-Poliklinisch Instituts der Universität Leipzig; and Prof. Dr. WOLFGANG ROSENTHAL, A.O. Prof. der Chirurgie an der Universität Leipzig. Royal 8vo. Pp. 444 + xv, with 501 illustrations. 1930. Leipzig: Georg Thieme. Paper covers, M. 24; bound, M. 26.

THIS volume deals fully with most aspects of surgery of the mouth, jaws, and face, and forms a useful book of reference to the subject for the general surgeon, who for the most part suffers from the disadvantage of meeting with individual cases from this somewhat heterogeneous group at long intervals.

Practical details of local anæsthesia and operations, many of them of a plastic nature, are gone into carefully, and a very large number of excellent illustrations add greatly to the value and interest of the book. The section on epithelioma of the mouth and tongue has not received the attention which its importance would seem to warrant, and no attempt is made to deal with the more recent development of radium therapy in this group of cases. With this exception the book sets out very fully the pathological, clinical, and operative aspects of this interesting group of cases.

The Student's Handbook of Surgical Operations. By Sir FREDERICK TREVES, Bart., G.C.V.O., C.B., LL.D., F.R.C.S., revised by CECIL P. G. WAKELEY, F.R.C.S. (Eng.), F.R.S. (Edin.), Surgeon to King's College Hospital, etc. Fifth edition. Crown 8vo. Pp. 535 + xi, with 190 illustrations. 1930. London: Cassell & Co. Ltd. 10s. 6d. net.

THIS very solid little book has now reached its fifth edition. The first edition was reprinted no fewer than eight times, the second edition three, and the third edition four times. The book has existed for thirty-eight years—nothing could be more loquent of the esteem in which it has been held by generations of medical students. Mr. Wakeley has abundantly justified the burden laid upon him in revising a work written by that facile master, Sir Frederick Treves, and nurtured for so many years by Jonathan Hutchinson.

The book contains a detailed account of practically every operation on vessels, nerves, tendons, etc.; in rather less detail it recites operations on bones and joints. Of plastic surgery, mouth and neck surgery, head, spine, and chest operations, there is an adequate account. The genito-urinary system is not forgotten, whilst modern treatment by radium and the cure of varicose veins by injection are carefully described. Operations on the rectum for hæmorrhoids and fistula are treated as fully as such a small book will allow. Abdominal operations and those performed for hernia are all remarkably well described.

The danger in a book of this magnitude is that the author, in a laudable effort to include everything, inevitably makes the book too big—this has been very skilfully avoided; enough has been included, but not too much. Those who are called upon to practice the major abdominal operations would naturally seek much bigger works for their guidance. We who have been brought up on this little book have grown to love it and to be very jealous of its future. We feel it is safe in Mr. Wakeley's hands, and we very strongly recommend the work to our successors. The binding, the paper, the printing, and the quality of the illustrations are excellent.

Die Chirurgie. A System of Surgery. Edited by Profs. M. KIRSCHNER (Tübingen), and O. NORDMANN (Berlin). Fasc. 29 (Vol. II). Royal 8vo. Pp. 1829–2028, with 98 illustrations. 1930. Berlin and Vienna: Urban & Schwarzenberg. RM. 16.

THIS excellent section on operations on bone and joints by Dr. A. Winkelbauer, of Vienna, completes Volume II, and fills in the gaps to which attention was drawn in our review of Fasciculus 26. The technique of bone suture, bone-grafting, and resection of the long bones is fully described and well illustrated. The approaches to the various long bones are presented rather briefly, and would have gained if accompanied by illustrations showing exact anatomical details. The exposure of the major joints is admirably described, especially the operations on the hip-joint, which contain a wealth of useful information. It is interesting to note the absence

of any reference to the work of Smith-Petersen in adopting and popularizing the method of approach to the hip originated by Sprengel. In removal of the internal semilunar cartilage, division of the internal lateral ligaments is still regarded as permissible, in spite of the growing tendency amongst Austrian and German surgeons to follow British technique in operations for internal derangements of the knee-joint.

The illustrations are bold and well chosen, although many are diagrammatic rather than anatomical. In the comprehensive bibliography British and American authorities are well represented.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Emergency Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London. Volume I. Abdomen and Pelvis. Medium 8vo. Pp. 380 + xx, with 324 illustrations, some of which are in colour. 1930. Bristol: John Wright & Sons Ltd. 25s. net.

A Treatise on Orthopædic Surgery. By ROYAL WHITMAN, M.D., M.R.C.S., F.A.C.S., Consultant to the Hospital for the Ruptured and Crippled, to St. Giles' and St. John's Guild Hospitals, etc. Ninth edition, thoroughly revised, Large 8vo. Pp. 1085 + xii, with 981 engravings. 1930. London: Henry Kimpton. 45s. net.

Diseases of the Chest. By W. J. FENTON, M.D., F.R.C.P., Consulting Physician, Charing Cross Hospital; Physician, Hospital for Consumption and Diseases of the Chest, Brompton, etc., and L. S. T. BURRELL, M.D., F.R.C.P., Physician, Royal Free Hospital, and Hospital for Consumption and Diseases of the Chest, Brompton, etc. Post 8vo. Pp. 384. Illustrated. 1930. London: Jonathan Cape Ltd. 15s. net.

A Text-book of the Surgical Dyspepsias. By A. J. WALTON, M.S., M.B., B.Sc. (Lond), F.R.C.S., Surgeon, London Hospital; Late Hunterian Professor, Royal College of Surgeons. Second edition. Demy 8vo. Pp. 720 + viii, with 286 illustrations in the text and 2 plates. 1930. London: Edward Arnold & Co. 42s. net.

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